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INTERNATIONAL ASSEMBLY

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Inter-State Post Graduate Medical Association of North America

CHICAGO ASSEMBLY

CHICAGO, ILLINOIS

October 26, 27, 28, 29, 1943

Published by
Inter-State Post Graduate Medical Association
of North America
Dr. Arthur G. Sullivan, Editor

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DIAGNOSTIC OTOLARYNGOLOGICAL CLINIC

Diagnostic Cases of the Lung and Esophagus

By ELMER W. HAGENS, M. D.
Assistant Professor of Otolaryngology, Northwestern University Medical School,
Chicago, Illinois

The cases to be presented can be divided into two groups. In the first group are four patients illustrating the value and importance of cooperation between departments. The internist, roentgenologist, endoscopist and chest surgeon are necessary in the solution of these cases.

Case 1 is Mr. W. S., aged 35, who complained of difficulty and pain in swallowing. The patient stated the symptoms began six months previously and that solid food would lodge part way down the esophagus. The pain was similar to a "heart burn," intermittent at first, but now for three weeks quite constant. Physical examination revealed only markedly enlarged tonsils. X-rays by Dr. James Case revealed a partial compression of the esophagus from the left side at about the level of the 7-8 dorsal vertebrae. There appeared to be an extra-esophageal tumor mass at this site approximately 4 cm. in diameter and in connection with the cardiovascular shadow rather than with the lung root. As to the exact nature of the mass Dr. Case could not say, but it might be glandular. Dr. Horrall, who was in charge of the patient, referred him to me for question of esophagoscopy. On examination with the esophagoscope at Wesley Memorial Hospital I found the left wall of the esophagus pushed in considerably, at the level seen on X-ray. However, the scope easily pushed the wall back, there was no pulsation or heaviness, and no change in the mucosa. Dr. Jerome Head was asked to see the patient and advised exploration, believing that we were dealing with a benign tumor, outside the esophagus. At operation a gland was found at the 7-8 dorsal vertebrae and removed. The patient recovered from the operation and at the present time (three months later) states he feels well and has no symptoms. Study of the gland by Dr. Strausser reveals chronic tuberculous lymphadenitis. X-ray study and barium fluoroscopy post-operatively by Dr. Case reveals normal findings and no evidence of the previous deformity.

Case 2 is Mr. T. G., aged 21 years. At the age of 13, in 1934, the patient had a cold and pneumonia, followed by pleurisy. Thoracentesis was unsuccessful, but several fistulous sinuses developed and drained for some months. The boy had enjoyed fairly good health prior to this time, but following the pneumonia and pleurisy he continued to have cough and expectoration, 1-2 ounces a day. Examination with lipiodol and X-ray revealed bronchiectasis of the left lower lobe. The patient was operated on by Dr. Jerome Head and the left lower lobe removed. Lipiodol injection on recovery in 1936 showed the bronchial stump and a slight involvement of the left upper lobe bronchi. The patient's left upper lobe apparently reacted unfavorably to the lipiodol, and he developed more secretion than previously. Colds have occcurred fairly often up to the present time. In 1938 the boy had a right upper lobe pneumonia and in 1940 a bronchopneumonia. During considerable of the time between 1936 and the present, 1943, the patient has been on a farm and has been given vitamins, iron therapy, and the best diet possible. The patient's general condition has been such that most of the time the medical men have advised conservative treatment, although in 1939 Dr. Head advised removal of the remaining left upper lobe; and in 1941 the Medical Department of the Northwestern University Medical School Clinic under whose management he has been, also advised considering surgery. Lipiodol injection in 1941 showed fairly normal right middle and lower lobes, but the left upper lobe had become the seat of extensive bronchiectatic cavities. From 1941 to the present the patient has been bronchoscoped frequently, sometimes weekly. As a rule considerable foul discharge is aspirated, with no improvement except for short periods of time and for a few days after the procedure. In March 1943 the patient was in Wesley Hospital because of a severe pulmonary hemorrhage, which fortunately stopped on bed rest alone. Bronchoscopic examinations before and since recovery have never revealed the exact source of the bleeding. Cultures taken at bronchoscopic aspiration have shown many hemolytic streptococci and a fewer number of other bacteria. It would seem in this case that conservative management is not sufficient and that if surgery is at all possible it should be done. Dr. Head feels it should be considered, but it would be quite an undertaking. For the present bronchoscopic aspiration can be carried out.

Case 3 is Mrs. H. B., aged 61, who began to notice a slight cough, January 1943. The symptom increased and has persisted. Shortness of breath became noticeable, but no chest pain. There has been no loss in weight. The sputum was scanty, never blood tinged or rusty. Examination revealed marked diminution of expansion of the left chest, dullness of the lower one-half of that side posteriorly, and weak, distant breath sounds. X-ray of the chest showed a soft increase in density of the left hilar shadow, left diaphragm difficult to visualize, and on lateral projection a shadow density in the left hilum. The findings were compatible with atelectasis in the left lower lobe and a tumor mass or infiltration of a gland. Dr. Jerome Head asked me to bronchoscope the patient, and on July 8, 1943 this was done. The carina was very broad and the left main bronchus extremely narrow at a point opposite the orifice of the left upper lobe bronchus. The bronchial wall was heavy, indurated, reddened and bled easily. Biopsy was taken from the medial-posterior wall, even though no fungations were seen. Further view of the left bronchus was impossible. The biopsy sections, according to Dr. Stausser at the Wesley Hospital, revealed an adeno-carcinoma. On July 14, 1943 Dr. Head explored the left chest, finding a diffuse matting of the tissues, definitely inoperable. At present the patient feels well although there is greater consolidation in the left chest. No treatment has been given the patient.

Case 4 is Mrs. R. G., aged 42. The patient states she developed a cough eleven years ago during a pregnancy, which diminished considerably after delivery. However, occasional cough has been noted for the past ten years. Thyroid operation was performed sixteen years ago. During the last eight months the cough has been much worse, being paroxysmal, with a whooping character. Lately the condition has been present daily, attacks lasting one-half hour. No blood has been noted, but vomiting has followed some of the paroxysms. There has been no fever. Various drugs taken have not given relief. She has not lost weight, in fact there has been a gain in the past several months. Hoarseness has been present frequently, but there was no history of dyspnea, hay fever, asthma, or dysphagia. General medical examination was essentially negative and X-rays of the chest likewise. As a result, Dr. Earl Zaus asked me to see the patient. Examination showed infection of the nasal antra and paralysis of the right vocal cord. Either or both of these conditions can produce cough and yet they would hardly explain the severe paroxysms. As a final check bronchoscopy was carried out and the findings were essentially negative. However, after several days, the cough disappeared and has been absent now for five months except for one short paroxysm last month. It is difficult to explain exactly what the mechanism has been in this case.

The second group of patients consists of three foreign body cases with instructive findings.

Case 5 is Baby R. R., aged two and one-half months. The baby had developed a stridor, inspiratory and expiratory, for one week following a previous upper respiratory infection. Treatment on the basis of an infection failing, Dr. Paul Lyons was consulted who believed there was likely a mechanical interference with tracheal breathing. A harsh stridor was heard but no rales. X-rays were taken which showed a toy ring in the upper esophagus, in such a position that the wire part pressed anteriorly against the posterior wall of the trachea. An older brother admitted that he had put it in the baby's mouth. At esophagoscopy the ring was visualized, but it rapidly moved into the stomach on attempts to grasp it. The procedure was terminated and the baby quickly recovered. At X-ray examination five days later, the ring was not in the child's body. In this case an esophageal foreign body produced only respiratory symptoms.

Case 6 is that of H. B., aged 10 years. The boy began coughing in August 1942. In October he was taken to St. Francis Hospital, Blue Island, with a pneumonia-like attack and with considerable fever. However on X-ray of the chest, in addition to decreased transparency of the left lower lung field, a metal density was found in the lower end of the left lower lobe bronchus. The boy then admitted accidently aspirating two months previously the metal wing of a toy airplane which he had placed in his mouth. Following recovery of the acute respiratory attack the foreign body was removed with the aid of the bi-plane fluoroscope. Coughing stopped almost immediately after bronchoscopy, and the boy rapidly recovered. In this case an unsuspected foreign body caused chronic cough and symptoms similar to pneumonia, but was fortunately opaque to the X-ray and therefore the true diagnosis could be made.

Case 7 is that of Mr. H. D., aged 40 years, who "choked" while eating chicken. Constant pain opposite the level of the third rib, anteriorly, and worse on swallowing, was noted. Dr. Behr referred the patient for question of foreign body. The patient was sent to Wesley Memorial Hospital and immediately given a hypo of morphine. In the morning X-rays of the chest and careful study of the esophagus with barium were done, but no evidence of a bone could be found. The patient felt well and believed the bone had gone on into the stomach. He was allowed to go home, but told to return if symptoms recurred. Five days later the patient reported that severe pain, as before, had developed. Esophagoscopy was carried out and a large bone, broken and in a V shape, 1¼ inches long, and lying crosswise was removed from the middle of the esophagus. There was considerable odor, some pus, inflammation and slight bleeding. The patient felt relieved immediately and recovered in a few days. In this case the patient's lack of distress and the negative X-rays findings led to delay in removal of a large bone in the esophagus.

DIAGNOSTIC UROLOGICAL CLINIC

Hormonal Therapy in the Treatment of Carcinoma of the Prostate

By NORRIS J. HECKEL, M. D.
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Statistics show that carcinoma of the prostate affects around five per cent of all men who reach the age of sixty years. Intensive efforts have been made to treat this disease successfully. However, those who have observed a considerable number of patients with advanced carcinoma of the prostate have found little encouragement from any method of treatment.

I think it is agreed by all urological surgeons that in early carcinoma of the prostate, that is, where the disease is confined within the limits of the gland itself, radical perineal prostatectomy is the proper procedure. Unfortunately, this group of patients is very much in the minority (not over five per cent) as compared with the group in whom the disease has extended beyond the borders of the gland by the time the patient presents himself to the physician for the relief of his symptoms.

The reason for this is quite obvious. Early carcinoma of the prostate, like carcinoma in many other organs, is a silent disease. It is only when the symptoms of urinary obstruction develop or pain from metastases occurs that the patient seeks relief.

The diagnosis of carcinoma of the prostate in the majority of patients is relatively easy to make, but at times it may be extremely difficult and must depend upon a biopsy. However, in a patient past fifty-five years of age whose complaints are difficulty of urination, backache and leg pain and whose rectal examination reveals a hard, nodular, infiltrated prostate, most certainly carcinoma is present. I know of no other disease of the prostate that produces such hardness or firmness as does carcinoma.

I should like to mention very briefly a new laboratory procedure that has been used as an aid in the diagnosis of carcinoma of the prostate gland with metastases. As you know, there are times when changes in the bones from Paget's disease and metastases from prostatic carcinoma may be difficult to distinguish. This laboratory procedure is an aid in making this differential diagnosis, and it also can be used as an index of progress in the use of endocrine therapy. This new method deals with the estimation of certain enzymes that occur in the body tissues, which are known as the acid and the alkaline phosphatases. There are obtained from the blood serum.

Acid phosphatase, it is said, can be increased in only one disease—carcinoma of the prostate gland with metastases. However, a patient may have an early prostatic carcinoma and the acid phosphatase still be normal.

Alkaline phosphatase also may be increased in this disease. However, there are diseases of the bone that will produce an elevation of the alkaline phosphatase, such as Paget's disease and osteogenic sarcoma. It follows, therefore, that an elevation of this enzyme is not specific for prostatic carcinoma.

In this discussion, I should like to limit my remarks to the treatment of advanced prostatic cancer.

The endocrine treatment for the relief of prostatic disease is not a new development. It has long been known, for instance, that there is a direct relationship and inter-relationship between the glands of internal secretion, especially the pituitary and the gonads and the function of the prostate gland. As a matter of fact, it was at about the turn of the Twentieth Century that castration was carried out for so-called benign prostatic hypertrophy. However, this was soon discarded. More recently, largely due to the experimental and clinical investigations of Dr. Charles B. Huggins of the University of Chicago, the endocrine treatment of prostatic carcinoma has been widely accepted. In general, the rationale for this form of therapy is as follows:

(1) Cancer of the prostate is often extremely sensitive to androgens or the male sex hormone, which, as is known, is manufactured mostly by the interstitial cells of the testes.

(2) The presence of androgenic hormone in the body activates the growth of prostatic cancer.

(3) The inactivation of the androgens brings about a retrogression of the growth of prostatic cancer. This retrogression can be accomplished in two

ways: (a) by castration; (b) by the administration of estrogens.

There is a difference of opinion as to whether or not these two forms of therapy will accomplish identical results. It is well known that in experimental animals castration increases the gonadotropic activity of the anterior lobe of the pituitary. The production of the male sex hormone is in turn under the the influence of the gonadotropic activity of the pituitary. It is also thought that other glands of internal secretion, notably the adrenals, can manufacture male sex hormone. It can be seen, therefore, that castration might not inactivate all of the male sex hormone that is produced in the body.

On the other hand, the administration of estrogen is believed to inactivate the production of the androgens by depressing the gonadotropic activity of the pituitary.

With these brief remarks, I should like to demonstrate a few patients whom we have had under observation for a moderate length of time. They have all had one form of therapy—the oral administration of diethylstilbestrol, which, you know, is a synthetic preparation.

Patient number one, 63 years of age, was first seen in December, 1942. He presented all of the symptoms of advanced prostatic carcinoma. He had lost sixty pounds in weight, was anemic, and could walk only with great difficulty. He suffered from a great deal of pain which required constant medication for relief.

Two years previously he had had a transurethral resection. The histological report was adenocarcinoma Grade III. I will not burden you with normal findings. The rectal examination revealed the prostate to be hard, nodular, and infiltrated; the borders could not be sharply defined. The x-ray pictures showed an extensive metastases to the spine, pelvic bones, and the ribs. He was placed on 10 mg. of diethyl-stilbestrol a day. I am well aware of the fact that 1 mg. a day of this drug is supposed to be enough to inhibit the production of androgens. However, in this particular patient, I decided to use much larger doses, and he was kept on 10 mg. a day for six months.

In about two weeks after this form of treatment was instituted, the patient's appetite began to improve, his weight increased, and his pain became less. This improvement has continued up to the present time.

As you can see, he appears to be in excellent condition; he can walk; there is no more pain; there is a gain of twenty-eight pounds in weight; he sleeps the night through without medication. The prostate is still hard, however, and nodular, but it is not as hard and infiltrated as it was before treatment was started.

I shall mention a few of the complications that we have noticed in using this form of therapy: they are really not very bad, but at times may be a little annoying to the patient. All the patients become impotent and sterile. The majority of them develop marked tenderness and fullness of the breasts and their nipples enlarge. Occasionally, leg cramps will develop. No one in our series has developed jaundice or a serious gastrointestinal upset. As yet, no one knows how long the patient should be treated with this form of therapy. In my opinion, the treatment should be continued indefinitely unless complications develop.

The next patient I saw first in May, 1941. He was at that time 61 years of age. Several transurethral resections had been done elsewhere. When I saw him, he had an acute retention, had lost weight and was anemic. The rectal examination revealed a large infiltrated mass on the right side of the prostate gland.

An indwelling urethral catheter was inserted, and later a transurethral resection was done. Histological studies of the prostate showed adenocarcinoma Grade III.

Diethyl-stilbestrol was given in June, 1941, 1 mg. t.i.d., and this has been continued more or less ever since. I say "more or less" because he is the patient who developed marked tenderness and fullness of the breasts, and there were periods of time when the hormone was discontinued.

At the present time, as you can see, he is in very good condition. He can work eight hours a day, has gained some weight, and the appetite is good. There is still a mild secondary anemia. He gets up only once a night and has no trouble with the urinary flow. Upon rectal examination, one can still feel the hardness in the prostate. However, it has diminished a great deal from the original findings.

The next patient was 75 years of age when first seen in 1942. At that time he presented symptoms of prostatic disease and he could not completely empty the bladder. The rectal examination revealed a hard, enlarged, nodular prostate. In September, 1942, a transurethral resection was done. The histological studies showed adenocarcinoma Grade III. Shortly after, he was placed on diethyl-stilbestrol, 1 mg., three times a day, and has been continued on that since.

The patient states he feels fine and is again able to carry on his job and works eight hours a day running an elevator. Although there has not been much gain in weight and he still gets up two or three times at night, his urine is crystal clear and in general I would say that he is holding his own.

These last two patients did not have as large a dose of the drug as the first one. I do not know, nor do I think anyone else knows, just what is the exact dosage. It would seem (and this is purely a "hunch") that the more extensive the carcinoma, the larger the dose; but this may not be true.

The next patient, whom I have had under observation for a long period, was seen for the first time in 1941 and at that time he was 59 years of age. Like the first patient presented, he had a typical syndrome of advanced prostatic disease—pain, secondary anemia, loss of weight. The prostate was hard, nodular, and infiltrated.

Because of the marked retention of urine, a transurethral resection was done, and I will show you histological sections of the prostate before and after therapy. The patient was placed on 3 mg. of diethylstilbestrol, and then the dose was rapidly increased up to 12 mg. a day. He was kept on this for approximately four or five months, and then the dose was dropped until he was getting 3 mg. a day. He has been on therapy since March, 1941.

He had extensive metastases to the spine and the pelvic bones, and these changes are still present.

Now, as you can see, the patient is a very healthy-looking person. He has gained weight, his appetite has increased, and he has no pain.

(Slide) Here is the histological section of the prostate gland on the last patient. It shows a typical adenocarcinoma.

(Slide) This shows the changes that have occurred in the prostate gland after he had about 1,500 mg. of diethyl-stilbestrol. You can see the extensive alterations in the cells characterized by retrogressive changes and marked vacuole formation.

(Slide) I said previously that these patients become impotent and sterile. Here is a lantern slide representing the total number of spermatozoa before the patient was placed on hormonal therapy. You can see that following therapy, there was a marked drop in the number of spermatozoa until he became completely sterile. The reason for this is the marked alterations that occur in the seminiferous tubules. On my left is a biopsy before treatment showing the normal tubules; here is a biopsy after estrogenic therapy, showing the marked retrogressive changes that occur in the tubules. This condition, I might add, can also be produced by the administration of androgens, although they do not make the patient impotent, nor does it dry up the seminal fluid.

Conclusions

1. It would seem that the clinical results which follow the oral administration of diethyl-stilbestrol are as great as those following surgical castration.

2. From a research viewpoint, it seems well demonstrated that cancer of the prostate may be influenced by changes in the endocrine system. From a clinical standpoint, it would appear that patients with advanced carcinoma of the prostate gland may be made more comfortable and perhaps may live longer.

3. The time is yet too short to evaluate end results.

DIAGNOSTIC MEDICAL CLINIC

Diagnosis in Pulmonary Diseases

By KARL J. HENRICHSEN, M. D.
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While diagnosis of pulmonary tuberculosis is usually simple, we meet with many other conditions of the lungs either associated with tuberculosis, or non-tuberculous, where the differential diagnosis may tax our intellectual facilities to the utmost.

In institutions, particularly where a large number of patients enter every year, one meets with many such conditions. Usually diagnosis of tuberculosis has been made, and in a greater number of cases correctly made. However, in some cases where a patient is admitted as a tuberculous suspect or for observation for other conditions simulating tuberculosis, particularly where the cardinal symptoms are absent or symptom predominating not characteristic in tuberculosis, we are called upon to differentiate.

Of the more common complaints in many such cases, is pain in the chest more or less severe. A patient with tuberculosis may complain of pain or soreness in the chest but usually not a severe persistent pain, it makes us think of other conditions, then if we find sputum negative for tubercle bacillus and perhaps negative tuberculin test, we can be reasonably sure that we are not dealing with tuberculosis. X-ray will usually give valuable information in all such cases.

Hemorrhage occurs in tuberculosis; it may occur in bronchiectasis; in abscess of the lung; in malignant diseases; in the mycosis and in passive congestion due to cardiac diseases. Then again, if sputum and the Mantoux test is negative, we must look for other conditions than tuberculosis.

Cough and expectoration is characteristic in tuberculosis and is found in most any other disease of the lung, even the common cold. The sputum findings are often of definite diagnostic value, particularly in tuberculosis, if positive; negative sputum means nothing. In lung cancer, cancer cells or sometimes a small particle of cancerous tissue may be brought up and be a valuable aid in establishing a definite diagnosis. Other organisms should be carefully looked for by culture or other methods available.

Careful clinical observation and study of each individual case is important and is possible only when the patient is confined to a hospital or institution with adequate facilities for work-up by X-ray, bronchoscopy and laboratory, with facilities to study biopsy specimens. This is necessary to make a correct diagnosis in any doubtful case that we may meet with.

Of the more common condition of the lungs besides tuberculosis, is malignant tumors, carcinoma; silicosis or silico-tuberculosis; cystic disease of the lung; Friedlanders infection; sarcoid; lung abscess; chronic passive congestion; bronchiectasis, and blastomycosis.

In differential diagnosis we must distinguish between the presence or absence of clinically active re-infection type of tuberculosis. This problem is usually presented to us in people who have been in contact with pulmonary tuberculosis. They may have a few slight symptoms which they exaggerate very astutely in such a way as to simulate pulmonary tuberculosis. However, tuberculosis may be ruled out by a careful history, physical examination, X-ray, repeated sputum and blood test, and a graded Mantoux reaction used down to 1 in 10. If negative, we may assure the patient that he has no clinically significant tuberculosis. If the results of such thorough work-up are negative, the patient is greatly aided in overcoming his symptoms by the thoroughness with which the work was done.

Another differential diagnosis is usually simple, but may be difficult. In every case of pulmonary tuberculosis we may have to be on the alert to discover an associated pulmonary or general condition which may alter the prognosis and treatment in a greater or lesser degree, as diabetes, which is commonly associated with tuberculosis.

Carcinoma of the lung is not uncommonly associated with tuberculosis and one may mask the other. Here severe and continuous pain is usually the predominating symptom, and a disproportionate amount of loss of weight for the type and extent of a tuberculosis lesion.

Cancer of the lung is being diagnosed much more often than it has in the past. It is confused because of the cough, hemoptysis, loss of weight and X-ray shadows which are usually confined to one lung.

Age helps a little in differentiating these cases, most of them occurring after the age of forty. Pain is a much more prominent symptom in these cases and appears in practically all cases at some time or other. The loss of weight in these cases tends to be continuous although it may remain stationary at times. We do not see the rapid response to treatment as shown by improved nutrition. Physical signs do not often help although a unilateral area of absent or suppressed breath-sounds must be investigated. If pleural exudate is found, aspiration from such a chest usually reveals a bloody fluid. Careful examination of this fluid may reveal cancer cells. X-ray shadows are unilateral and have definite hazy borders, rather than the mottling effect of tuberculous involvement. It simulates the pneumonic type of tuberculous disease.

When fluid is present even to the level of the 1st rib, there is not the shifting of the mediastinum which is seen in tuberculous effusions. Bronchoscopy will reveal obstructive tumors in a considerable proportion of these cases and associated with biopsy clinches diagnosis. Lipiodols will occasionally demonstrate definite bronchial obstruction without resort to the more difficult procedure of bronchoscopy. Diagnostic pneumothorax outlines tumor densities and rules out pleural effusions causing diffuse haziness of a whole hemithorax.

Chronic passive congestion of the lungs is a condition which may be caused by any change which obstructs the circulation of the pulmonary circuit. Mitral-stenosis is the most common etiological factor that may cause this condition but it may be caused by aortic lesions, aneurisms and mediastinal tumors such as lymphosarcoma or Hodgkins Disease. They may present all symptoms of pulmonary tuberculosis including hemoptysis. The history usually reveals an antecedent rheumatic infection and careful physical examination usually reveals a mitral lesion of the stenotic type. The X-rays in these cases show the typical mitral configuration of the heart widening of the hilus shadows and shadow strands radiating out into the parenchyma are increased in size and density. The differential point here is that these shadows are approximately equal in all three fields; moreover, any one of these shadows if followed towards its termination shows a progressive decrease in size and density.

Lung Abscess

Lung abscess may be confused because of the cough, expectoration and hemoptysis with toxic manifestations. There is in the majority of cases of lung abscess a history of a previous tonsilar or nasal operation or other general operations immediately preceding the onset of symptoms. In those that do not fall in this category there will usually be a preceding pneumonia of the area involved. The symptoms are more violent, the patient is much more acutely ill than the tuberculous patient with the same fever curve. The sputum is foul, separates in three layers and contains no tubercle bacilli.

(Slide) This slide is the X-ray of a man 60 years of age. He was admitted with diagnosis of far advanced pulmonary tuberculosis and positive sputum. After two years in the sanitarium he was discharged on his own request, but did not get along and was readmitted one month later. (Slide) The X-ray now shows a large tumor mass extending from the hilum to the periphery of the right lung.

Diagnosis is made of malignant tumor, based on the unusual X-ray shadow.

The patient expired and autopsy shows healed bilateral pulmonary tuberculosis, and primary bronchogenic carcinoma of the right main bronchus.

(Slide) This is a man 44 years of age. Complains of occasional violent pain on the right side of the chest on taking a deep breath; he has moderate cough, mostly at night on retiring; expectoration, slight but sometimes bloody; fatigue for the past six months.

Laboratory reports: Sputum negative for tuberculosis; on daily examination made for cancer cells is found a small mass 5 x 10 mm. in size, which on microscopic examination is seen consisting of a vascular stratified epithelium structure with much anaplasia, many mitotic figures and occasional horn pearls.

Bronchoscopy gives no information.

X-ray shows an area of haziness extending from the right hilum outward to the periphery and upwards, and the next X-ray (slide) taken six months later shows much more extensive involvement.

Diagnosis: Bronchogenic carcinoma.

(Slide) This is a man 64 years of age, a roofer who has been doing hard, outside labor for many years. In April he began having pain in the left side of his head traveling to his left shoulder and back. This pain became excruciating. He became short of breath and developed a cough. He consulted his physician and was advised to enter the hospital. He was admitted to the Sanitarium.

On admission he complained of cough, severe at times; pain in left chest and his back all the time, and at times very severe. Dyspnea was persistent. He had lost 50 pounds in weight since the onset.

Physical examination of the chest revealed very little. The chest was barrel shaped. There was impaired resonance in both uppers and diminished breath-sounds throughout; no moisture elicited. Diagnosis was deferred. Consider tumor or tuberculosis.

Laboratory reports: Negative sputum to culture on animal inoculation.

X-ray shows an increase in the bronchial markings on the right lung. On the left is a large annular shadow of the hilus, appears as a large cavity. There are several small cavities extending upwards into the apex.

Patient expired in a profuse hemorrhage after two months stay in the hospital.

Autopsy revealed carcinomatous abscess in the base of the left upper lung lobe and the apex of the left lower. There was metastatic carcinoma of the lymph nodes and around the lung hilum and the aorta.

(Slide) Tuberculosis with silicosis presents a difficult problem in men with an industrial history. Here the physical examination may reveal exceptionally poor expansion, marked suppressed breath-sounds, especially over the midfields and sometimes over the entire chest. The X-ray may be suggestive of tuberculosis, but shows in addition the widened hilum, increased shadow strands and nodular mottling in the mid-lung field.

A man 53 years of age gives a history of having been working on grind-stone for several years and factory work.

Complains of severe dyspnea, severe cough and expectoration, out of proportion to the amount of involvement if tuberculosis, tired and weak.

Sputum is positive for tuberculosis.

Diagnosis: Silico tuberculosis.

Diagnosis of silicosis is made by the history of occupation working on grind-stone, and by X-ray.

(Slide) A woman, 37 years of age, complains of having a cough, some expectoration, feels tired, and dyspnea for about a year. Her doctor told her she had some congestion on her right lung and that the thyroid gland was causing her trouble. She was admitted for observation. Physical examination revealed diminished expansion on the right side, persistent rales and suggestion of cavitation on upper one-half bilaterally. Sputum at first seems positive for tuberculosis, but after repeated and thorough study it was found to be non-acid fast organisms.

Mantoux was completely negative.

Skin manifestations: a purplish red endurated area size of a quarter was found on the right side of her forehead, no scaling or vesicles; another is found at the vertex of the head same color but covered with scales. Biopsy of these lesions reveals small granulomatous nodules of loose epitheloid cells and occasional lymphocyte, several abortive giant cells, no caseation. A picture of Sarcoid.

A woman, 51 years of age, housewife, onset in January, 1940, with a chest cold and diagnosis of pneumonia, but she has not been well since.

She complains of cough, slight expectoration, is weak and tired. She has lost 15 pounds in weight. Admitted for observation. Examination reveals suppressed breath-sounds and rales over the right and upper left. Sputum is negative to culture and animal inoculation. Mantoux is completely negative. After further study and observation in January, 1941, final diagnosis was made Sarcoid.

Next X-ray one year later shows more involvement on the left side.

This is a man 57 years of age, laborer, has been working in grain elevators for 35 years, handling various kind of grain.

He complains of feeling very tired. He has a moderate cough and some expectoration and about a year ago had some bloody sputum. He is getting more and more dyspneic for the past year; no loss of weight.

Physical examination of the chest reveals a well nourished man, emphysematous shaped chest, diminished expansion bilateral, slight impaired resonance at the apices. Suppressed breath-sounds everywhere; there is a few indefinite rales above the clavicle.

Laboratory reports: Persistent negative sputum to culture and animal inoculation.

X-ray shows infiltration on both upper fields characteristic of a moderate advanced tuberculosis. On the 10th rib, anterior, is seen a small area of rarefaction, 1 cm. in diameter. The rib is resected for biopsy and laboratory reports on cutting the rib on the rarefied area shows a golden yellow soft sharply defined nodule.

Microscopic study reveals that the nodule consists of large pale cells with granular fatty cytoplasm of a foamy appearance and with small round nuclei suggesting metastasis from a hypernephroid carcinoma.

On later studies final diagnosis of sarcoid was made.

Patient is a young woman 32 years of age, housewife, past history is essentially negative; normal pregnancy and delivery five years ago.

Onset with cough, becoming severe and persistent and not relieved by medicine. She was admitted to the Sanitarium for observation.

Complaints: Severe persistent cough, day and night expectoration, one ounce in 24 hours. Pain on right side after coughing; loss of weight and marked fatigue.

Physical findings: Chest: Impaired expansion on right; decreased vocal fremitus upper right; dullness on upper right to sixth rib posteriorly; suppressed breath-sounds in upper half and posttussive rales on right; left negative.

Laboratory reports: Wasserman negative; basal metabolism plus 22. Sputum and stomach wash negative for tubercle bacillus to culture and animal inoculation. Mantoux positive 1-10,000. Bronchoscopy negative.

Biopsy of a small gland removed from the anterior right chest wall shows foci of many Dorothy Reed cells, showing hyperchromatic irregular nuclei.

X-ray shows an irregular tumor-like mass extending from the mediastinum toward the periphery and from the apex to about the 9th rib on the right side; left is negative.

Diagnosis: Hodgkins Disease.

A woman, age 60, had pneumonia on right side in 1937. Was ill three weeks.

Onset in January, 1939, when she began having colds, three or four attacks, one after another, associated with some cough and temperature to 100 and 101. Moderate fatigue and pain in the right upper chest. She was treated with diathermy and other local measures. Symptoms and pain disappeared in a few days. The last attack occurred in October, 1939, at which time X-ray of chest was taken and showed atypical pathologic changes and what appeared to be an acute exudative process in the right base. Some rales were heard over the right side at that time. Patient was admitted to the sanitarium for observation and diagnosis.

Complained of cough in the morning; expectoration slight; pain in right upper chest, worse with breathing and on coughing.

Physical findings: Impaired expansion on the right side. Resonance impaired on right apex; breath-sounds are suppressed and a few mucous clicks are heard on the upper right lobe; left is negative.

X-ray taken on admission shows most of the basal process has cleared up in four weeks time.

Laboratory reports: Mantoux positive 1-1,000. Sputum negative for tuberculosis, is a greenish mucopurulent material. Direct smear; large number of gram negative incapsulated bacilli. Moderate number of fusiform bacilli and spirochetes. Twenty-four hour culture grams negative bacilli. Friedlanders Bacillus. Friedlander's Bacillus Infection.

Onset about February, 1943, very insidious with slight but constant cough, becoming more severe, fatigue and more tired toward evening. His doctor advised hospitalization, examination and X-ray revealed tuberculosis?

Admitted to the Sanitarium. Cough was severe and very troublesome. Expectoration at times was bloody; severe shortness of breath.

Physical examination reveals a fairly well nourished but very sick man.

Right lung there are diminished breath-sounds and on the left, tubular breathing suggesting cavitation on the upper third and moist rales after coughing from apex to base.

X-ray shows some mottled infiltration on the right side extending from the hilus out and upward to the periphery. Left side shows more extensive mottling extending from the hilus to the periphery and apex. This is not a typical picture of tuberculosis and this man is more acutely ill than we would expect from tuberculosis. Temperature 99 to 101; pulse 110 to 140.

Laboratory reports: Mantoux negative to 1 to 10. Sputum negative for tubercle bacillus.

Diagnosis: Carcinoma of left lung.

Patient expired two months after admission. Autopsy revealed Blastomycosis.

A man, tailor, age 50, admitted to the sanitarium on several occasions because of repeated hemorrhages. He was diagnosed as pulmonary tuberculosis by several different physicians because of the hemorrhages and the marked increase in shadow densities on X-ray. Mantoux tests were negative in 1 to 10 dilutions and the subcutaneous injection of 100 mgs. of O. T. gave no systemic or focal reaction. His X-ray in addition to the usual findings of a marked Passive Chronic Congestion of long standing, shows a typical falciform shadow indicating an Azygos Lobe.

This X-ray is typical of marked passive congestion of long-standing with considerable fibrosis of the lung parenchyma, evidenced by the diffuse mottling in the periphery. It might be confused with Miliary Tuberculosis as well as more chronic forms of pulmonary tuberculosis.

This is a young man 22 years of age who came to the Sanitarium as an observation case and for diagnosis.

History: He went through high school, had been working as shipping clerk, now as engraver and photographer. In January, 1938, while sitting still at his work, he began to feel short of breath, getting worse so he could not breathe except with great effort, standing or lying down. He remained in bed a few days,—still very dyspneic. The doctor was called, X-ray was taken, which revealed collapse of the right lung. He was hospitalized and an exploratory operation was done to look at the lung. Following this, more collapse occurred and continuous suction was instituted. The lung re-expanded in five to six weeks and he went back to work, still dyspneic. Then in January, 1941, he became more and more dyspneic. His doctor advised hospitalization and he was admitted to the Sanitarium. He complained besides the shortness of breath, of moderate severe cough at night and expectoration (12 grams in 24 hours) of a greenish material.

Physical examination revealed a slender young man, rather thin, weight about 115 pounds; impaired expansion and impaired resonance; harsh breath-sounds alternating with suppressed breath-sounds, and some fine rales after coughing, over the entire chest. Temperature—normal; pulse 80.

Laboratory reports: Wasserman and Kahn—negative; sputum—negative for tubercle bacillus to culture and animal inoculation; Mantoux—positive.

X-ray shows extensive fibroid infiltration over the entire lung field with numerous small excavations or possible cysts, more on the left lung. Bronchoscopy reveals nothing of diagnostic value.

Clinical diagnosis: Chronic cystic lung disease.

Those cases that present symptoms simulating tuberculosis because of bronchiectasis are usually of long duration often have a history of a previous pneumonia, influenza or silicosis and fibrous pleuritis or old empyema. Characteristically they are subject to periods of exacerbations which are usually ushered by acute upper respiratory infections and may be followed by remissions in which all the symptoms disappear for weeks or months at a time. The physical findings in these cases during the period of remission are very slight and may be entirely absent. Rales if present are usually found in the bases of one or both lungs. The sputum varies in amount being highest during the period of exacerbation at which time eight to sixteen ounces of three-layered material may be presented.

X-rays show very little in mild cases but studies with stereo plates may show some haziness of the cardiophrenic angles, a haziness of the cardiac borders and small annular shadows in the bases resembling a mesh-work of rings. In a case with these findings the matter is promptly cleared up by the use of lipiodol injections and X-ray studies made after this injection show definite sacular or cylindrical dilatations of the terminal bronchi with blunting of their ends. This may occur in any lobe including the upper lobe but in the majority of cases occurs in one or both lower lobes.

DIAGNOSTIC MEDICAL CLINIC

HEART DISEASE

By ROBERT S. BERGHOFF, M. D.
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May I say that it is a distinct pleasure and a privilege to present a clinic before this grand organization. I may say to you, too, that it has been a pleasure and a privilege to have acted as Chairman of your Clinics for this year.

I am to present a clinic for a half hour on heart disease, my specialty. I have selected as my subject coronary disease. I appreciate that it is a pretty ambitious idea to try to cover that type of subject in a half hour. I have chosen it deliberately for several reasons. In the first place, I think you will agree that to doctors, nurses, all of us who have to do with sickness and health, probably no type of heart disease is more intriguing, for various reasons. In the second place, coronary disease has a very personal appeal, it seems to me, after we get into our fifth and sixth and seventh decades of life. I don't want you to be too serious-minded about yourselves, but I call your attention to the fact that as we get into the fifth, sixth, seventh and eighth decades of life (and I have slipped past the fifty-year mark myself) two, possibly three, out of five of us become conscious of our coronaries, so this subject should have a very personal appeal to all of us.

I shall try in the next half hour to cover at least the highlights and highspots on this, to me, intriguing and interesting subject of coronary disease.

In the first place, let me call your attention to the fact that even though we encounter coronary disease in some of the earlier decades of life, the fourth decade, the third decade, even the second decade of life, in the teens (several investigators and clinicians, particularly in the East, have come out recently with a series of coronary patients based on arteriosclerosis, if you please, at the age of twelve, fourteen, fifteen, sixteen and eighteen, all verified after death), the vast bulk of coronary disease belongs to old people. Coronary disease is part and parcel of senile decay and it belongs in those later years of life.

Coronary disease, with the exception of those instances which are on a background of a hypertension or a valvular disease, are due to arteriosclerosis and more specifically they are due to arteriosclerosis of the coronary arteries; still more specifically they are due to atherosclerosis of the coronary arteries.

I call your attention to several interesting points. The coronary arteries, as you will remember, have their origin in the aorta near the cusps and the pressure of the constant closure of the aortic cusps exerts a pressure upon the intima of the coronary vessels, leading gradually to a thickening of this intima, and then to a roughening, paving the way for the superimposition upon that roughened intima of the coronary of the future thrombus. That probably is the most common mechanism for coronary thrombosis.

I brought with me three patients this morning to prove or bear out one rather interesting point that I should like to have you take with you at the end of this half hour. In my mind and in the minds of my associates, coronary disease is a relative situation; I mean it is not a matter of one man having it and another man not; it is a relative and it is a cyclic, a progressive, pathology.

My three patients here this morning are to bear out that point. Coronary disease due to atherosclerosis beginning in later life begins usually in the form of an angina pectoris, an angina of effort. The individual who is going to have his coronary occlusion and possibly have later on, if he lives long, his status angiosis or terminal stage of that disease has an angina pectoris first. By angina pectoris we have always understood, of course, that rather dramatic episode that you all know so well that I would not take up your time nor mine to describe clinically. By angina pectoris you and I understand clinically that episode which occurs very suddenly in people usually past fifty following physical effort or following mental excitation or following a gastrointestinal indiscretion.

Clinically that episode of angina pectoris consists of either a rather severe, short-lived, precordial pain with typical reference into the jaws or down the left arm or the right arm or both, implicating particularly the wrists, with severe, excruciating pain in the wrists, possibly going all the way to the finger tips; a short-lived, excruciating pain following physical effort or mental excitation, almost, always a causative factor of brief duration, completely and quickly relieved by coronary dilators, particularly nitroglycerin, or completely and quickly relieved in a very few moments by physical and postural rest with no drugs whatever. That is angina pectoris or angina of effort. We have always believed that the pain, or in the absence of pain the shortness of breath or the strangulation, had as its mechanism a spasm of the coronary trunks. I am not here to stick my chin out, figuratively, and recommend that we leave that theory in the discard entirely. Let's agree to this: The individual with his first stage, so-called, of angina pectoris at least has a beginning coronary pathology, a narrowing, either circumscribed or general, of his coronary trunk and because of that an insufficient coronary flow and again because of that a myocardial ischemia, and because of those sets of circumstances he has a typical anginal syndrome.

There are very good reasons that will occur to you as to why these three patients are not going to be examined, why even their X-rays and electrocardiographs cannot be passed around in a huge throng such as this, this morning, but I want them here as living examples or as impressive examples to you of the relativeness of coronary disease. For example, this first patient, Mr. Atherton, should represent to me and to you the individual with early coronary pathology in the guise of angina of effort.

May I say at this point when I am speaking as personally as I am about three patients sitting in front of me, I talk to them now for a moment and not to you, and I say that whatever I say about you should not be discouraging to you because as I look this throng of doctors over, who come from all over the country, beginning with myself at fifty-four, what symptoms you have I as yet have not had but may have next week or next month—it comes to all of us.

My first patient, this gentleman, gives a story of angina of effort a year ago, and he is sixty-nine years old. At the age of sixty-eight this first patient contracted or gave subjective evidence to himself of angina of effort through a severe, suddenly occurring precordial pain referred to his jaws and down his arms, associated with some dyspnea, quickly relieved by posture the first time, and the recurrences quickly relieved by nitroglycerin; no attack, the first or none since, ever associated with the symptoms and signs that you and I associate with a complete occlusion and infarction.

Up to a few decades ago angina pectoris, angina of effort, to us meant an individual who probably had a spasm of his sclerotic coronary artery and because of that spasm had an insufficient coronary flow reaching his heart muscle, which rebelled and caused pain and dyspnea.

Possibly we have not left the theory stage completely, but it is our best belief, the belief of those of us who have to do constantly, exclusively with cardiology, that the vast bulk of these individuals with so-called angina of effort—not all but the vast bulk—have their pain and their dyspnea not because of a spasm of a coronary trunk but they have their pain and their dyspnea as mute proof of the pre-existence of an occlusion of a coronary trunk. They have already had a closure possibly of a smaller tributary of one or the other coronary, but certified proof (and you know what I mean) is piling up that most of these individuals with angina of effort have as an explanatory background for their mechanism a previous occlusion with a mild and well healed infarction. That is the story of this gentleman.

We come to No. 2, Mr. Edward Wright, and he is sixty years old. To bear out my cycle, my progressiveness of coronary disease, my relativity, if you will, of coronary pathology, he then should exemplify a further step in the coronary circle or chain, and so he does. From his history he tells us that his situation began quite a long time ago with a typical angina pectoris, angina of effort. After repeated attacks of this angina of effort, this gentleman, Mr. Wright, age sixty, then sustained a typical acute occlusion with infarction. By that I mean and you mean that rather dramatic episode that is not very readily confused with the more simple type of so-called angina of effort. This gentleman, Mr. Wright, as all patients who have had a diagnosis of acute occlusion with infarction certified have either following physical effort or mental excitation or while at absolute rest with no apparent directly logical cause whatever, had a sudden, severe, prolonged precordial pain much like the pain of angina of effort except for a few important differentially diagnostic factors, to wit: their pain is of longer duration, hours, even days; it is relieved either not at all or only partially by the coronary dilators or by postural and complete physical and mental rest; but the differential diagnosis between angina pectoris of effort and acute occlusion of heart muscle invasion of considerable degree hinges clinically upon the picture that is so dramatic and which we have all seen and which we would have great difficulty in mistaking, to wit, again, the symptoms of shock. I mean the individual with acute occlusion and infarction, with his long drawnout, long sustained precordial pain, or, in the absence of pain, strangulation lasting for hours or days, has as associated symptoms shock, particularly sweating, profuse sweating, and not only physical manifestations of a very severe heart muscle invasion which you and I discover with our stethoscopes and with our fingers, but the subjective symptoms also of severe heart muscle invasion such as a terrific heart consciousness through an irregularity, through a feebleness, through a loss of tonus. The individual with this occlusion feels within himself that he is desperately ill and that it is not a short-lived business with him. He does not know about the outcome and neither do you nor I for the time being.

We then, through our physical examination, find a heart that actually has been mortally hit, with a rhythm that is vastly disturbed, a rate that is either increased or blocked off quickly, a very poor volume, and heart tones so distant we can heardly hear them.

The electrocardiograph, of course, can be and frequently is very indefinite but in the first instance of angina of effort gives plenty of corroborative evidence of a heart muscle invasion.

So far I have talked to you about angina of effort and acute coronary occlusion with infarction. The individual with acute coronary occlusion with infarction may (he doesn't have to but he may) after healing his infarcted area pretty thoroughly, as the years add a further toll through physical and mental strain, physical strain particularly upon this infarcted heart muscle, go into a situation where clinically he has practically always on the slightest effort evidence of an insufficient coronary flow through pain or dyspnea or both. The cycle is spreading, you see.

Mr. George Thomas is sixty-two. His story and his physical findings and his electrocardiographic evidence point to the fact that he has had his angina of effort and he has had his acute occlusion and that unless he is careful he gets himself into physical trouble with his heart.

It should be very plain why the last ten minutes of this discussion should be carried on without the presence of the patients. I am approaching the subject not only of management of these three cycles in coronary disease, but the prognosis as well.

What is the outlook or the prognosis of angina pectoris? You and I know the answer to that pretty well. The outlook is so much better than we used to consider it to be only a short decade ago that it is one of the few pleasures in cardiology. We get these nice individuals, men and women, in their fifth and sixth and seventh, aye, in their eighth decades of life, in their first stage of coronary atherosis, and through proper management we forestall the second stage or period, acute occlusion with infarction, not a year but five years or ten years, many times even twenty.

What is the outlook for the individual in the second chair with acute occlusion and infarction? Of course, he has had a more progressive pathology. His pathology has marched on. It is not a matter of years, necessarily; it is a matter of physical strain and a combination of other circumstances. His outlook, too, is so vastly improved over just two or three short decades ago that whereas twenty or thirty years ago we believed the mortality in the acute stage of occlusion with infarction was 12, 15, 18, 20 per cent, that prognosis has entirely changed and these people can be revamped and reeducated to the point where they can live not only a long time but live viable, useful, progressive lives. When we get into that situation, of course, the story is different. When our pathology has marched along so progressively and gone through the stage of angina of effort; gone through acute occlusion with infarction, and where our individual has reached a so-called status angiosis, where on the slightest physical effort or the slightest mental excitation or even sometimes simply dressing himself or herself there is pain and dyspnea, naturally the degree of pathology is so great that the outlook is very limited.

Just for fear that I might not be able to give you these few little things that I have jotted down which I consider the nubbins of this brief half hour on coronary disease, I will forbear the matter of treatment of these three types of coronary disease, because I could never finish them anyway in three or four minutes, and give you these points.

I apologize if I am too elementary, if most of you already know or have been impressed with these important points, but unless I review them with myself every once in a while I forget their importance.

(1) Most so-called angina of effort has as its pathological background a previous occlusion. That is dynamite; it is controversial and some cardiologists do not agree, but a lot of experimental proof is piling up.

(2) Coronary disease is a disease of senility and not a disease of youth, as the lay publications and the radio and even some of our medical publications would have us believe.

(3) It is due to arteriosclerosis, or the vast bulk of it is, and that in turn to an atherosclerosis.

(4) It is progressive and cyclic and relative.

(5) The pain or discomfort or strangulation in all cases is due to a varying degree of coronary insufficiency, which in turn carries less blood to the heart muscle than it needs and we have a myomalacia as the final background for the symptoms.

(6) The clinical diagnosis of acute infarction I have given you as associated with shock.

(7) In early life, the first segment of the left coronary is more frequently involved than any other portion of the coronary system.

(8) In later life the right coronary is as frequently occluded as the left.

(9) More sudden deaths are due to left coronary lesions in our experience and the experience in cardiac literature than to right.

(10) Paradoxically enough, anastomosis between the coronary vessels increases with age. When a man's heart is old, paradoxically he has a more rapid and complete anastomosis between his coronaries and develops for himself coronary circulation than does a young man. Nature is truly kind.

(11) I have already pointed out to you that you must not be confused if in your practice you encounter not too infrequently coronary occlusion with infarction, even on the background of arteriosclerosis and atherosclerosis in very youthful individuals in their teens and between twenty and thirty. They are exceptions to prove a rule but they are there.

(12) Finally, in youthful individuals pathologically the thrombus we encounter is usually a vascularized soft thrombus. In old people the thrombus is calcified, and in middle age we find a combination of the two.

My time is up and I know that I have rambled along in a desultory sort of fashion, for which I hope you will forgive me.

DIAGNOSTIC UROLOGICAL CLINIC

The Significance of Blood in the Urine

By HERMAN L. KRETSCHMER, M. D.
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As you all know this country is a great country for slogans. Every nationally advertised product has a slogan. Every candidate for public office has a slogan. I wish that every physician in this country would have a slogan on the significance of blood in the urine. I would like to present for your consideration whenever a patient consults you with blood in the urine, the following slogan: "The presence of blood in the urine means the presence of serious organic disease until the presence of serious organic disease has been ruled out." This would result in improving the quality of medical care because the patient would receive the benefit of early diagnosis instead of a late diagnosis. Apropos of this may I say that I believe the time is ripe to have a campaign on the importance of blood in the urine just as we have other health campaigns, such as the campaign for the early diagnosis of cancer and the campaign for a yearly physical examination.

It is to be remembered that when a patient has hematuria, the bleeding always stops which, in a measure, is rather unfortunate because it gives the patient and sometimes the physician a false sense of security. If the first attack of hematuria would continue without interruption, the patient would become exsanguinated and he would die. On the other hand, if the patient has taken some medicine on his own account or if he has received medication from his physician, then the cessation of bleeding is wrongly ascribed to the particular drug which was administered and this false sense of security leads only to delay.

About thirty years ago various designations were given to this group of cases, such as "essential hematuria," "renal purpura," "bleeding from normal kidneys," etc. At the present time these phrases are rarely, if ever, used and they only serve as a cloak for our ignorance or our inability to make an accurate diagnosis. The reason that these terms have disappeared from clinical nomenclature is due to the fact that at the present time, with our diagnostic armamentarium, the cause for the bleeding is determined in the largest number of instances. It seems to me that in the very small group in which no cause for bleeding can be determined, it would be much better to designate them as cases of bleeding of undetermined origin.

There still remains a very small group in whom, if complete urological study has been made, one cannot determine the cause for bleeding. Let me consider a few of the possibilities in this small group of patients. The patient may have had an attack of hematuria due to stone in the upper urinary tract. During the attack of hematuria, or subsequently, or unbeknown to himself, he passes the stone so that when the examination is made, no cause for the hematuria is found. Likewise the same situation may occur in a patient who has had infection; the patient may have had an acute colon bacillus or other bacterial infection which manifests itself in painless hematuria; the infection runs its course; then when the patient comes in for an examination nothing is found because, as I have just stated, the infection has run its course. Again, a patient may have a stone as the cause for his bleeding and the stone cannot be demonstrated with roentgen ray because the stone may have the same density as and be overlying one of the bony structures.

The causes of hematuria are many. When discussing the subject of hematuria with the students, I try to impress upon them the fact that the causes for bleeding are many and I like to compare them in number with the advertisement of the "57 Varieties." A good clinical rule to learn is one that places the causes of hematuria into two groups: the general causes and the urological causes. I shall not burden you with a recital of all of the different lesions that may cause hematuria; you are just as familiar with them as I am. On the other hand, although the number of causes of hematuria are many, from the practical point of view it is well to bear in mind that there are five common causes of hematuria, namely stone, tumor, tuberculosis, infection, and nephritis. If you bear them in mind, you will be impressed with the fact that each patient who bleeds has a serious lesion of some sort which demands a complete urological study.

I have selected four patients that illustrate some of the problems in differential diagnosis of this subject and who demonstrate the fact that the urologist must be a good general diagnostician and be familiar with the fact that, although the patient may bleed from the urinary tract, the cause for the bleeding may be located in distant parts of the body. With this in mind, I should like to present the histories of some patients with hematuria.

Case I. — Hematuria due to Streptococcus in the Nose and Throat

The patient, female, aged thirty-five, gave a history of painless hematuria. On two occasions the hematuria was associated with urgency and frequency. The patient stated that she had felt dizzy and experienced a few momentary chills two days before admission to the Presbyterian Hospital. The physical examination, as well as the blood pressure, pelvic examination, roentgen ray for stone, intravenous pyelograms, all were negative. The cystoscopic examination showed multiple areas of submucous hemorrhage scattered all over the bladder. Submucous hemorrhages distributed all over the bladder are very frequently associated with infection due to the streptococcus. This tentative diagnosis was made at the time of the cystoscopic examination. Bacteriological examination of the urine showed the presence of hemolytic streptococci. On the basis of the tentative diagnosis made at the time of the cystoscopic examination a search for the presence of a foci of infection elsewhere in the body was carried out. Cultures were made from the secretions of her tonsils and nasal pharynx and these cultures showed hemolytic streptococci. Therefore, it was perfectly obvious that the cause of the hematuria, in view of the fact that the urological examination showed no cause for the bleeding, was located in her nose and throat. She was referred to the otolaryngologist who removed her tonsils; and again, from her tonsils, the same type of organism was found in cultures. The infection in her naso-pharynx was cleared up with appropriate treatment.

Discussion: In this instance we were dealing with a patient who had a symptomless hematuria. There was nothing in the history to indicate that she had an infection in her tonsils or her naso-pharynx although she did admit having a mild sore throat once in a long time. It was of short duration and she paid little, if any, attention to it. In this type of lesion it is well known that the patients not only have submucous hemorrhages in the bladder which one can see with a cystoscope but hemorrhages are also found in the ureter and the kidney pelvis.

As I have stated, the cystoscopic picture was rather characteristic and it was because of the cystoscopic picture and because of the well recognized fact that this picture is often due to infection in teeth, tonsils, and sinuses, that the examination was at once directed to looking for foci of infection elsewhere. A recent interview with this patient revealed the fact that she has had no more bleeding, her urine is negative, and she is perfectly well. Therefore, by recognizing and clearing up the infection in her tonsils and throat, this patient was spared not only further attacks of hematuria, but possible other serious organic diseases.

Case II. — Hematuria due to Hypertension

The second patient whose history I would like to discuss with you was a man, aged fifty, who gave a history of painless hematuria which had occurred intermittently for two months. There was associated some nausea and vomiting, a little dysuria, and for one day he complained of pain in his back. The pain did not radiate as does the pain in renal colic. The general physical examination was negative except for the fact that the systolic pressure was 165, and the diastolic pressure was 100. Examination by roentgen ray, cystoscope, and pyelograms were also negative. Examination of the urine showed albumin 1+ but no casts and a few white cells. In this patient the only positive findings that we were able to demonstrate was that the man had hypertension. He was then referred to the medical service for treatment.

Discussion: I think it is very important to bear in mind the fact that the patient with hypertension may bleed and that the hematuria may be due to the hypertension. I would also like to stress the relationship between these two conditions which is often overlooked. I think we fail to recognize that the patient with hypertension may bleed and that the patient with hematuria may have hypertension and that a direct causal relationship between the two is at hand. On the other hand, the patient with hypertension may also have a stone in his kidney or ureter or bladder or a tumor of the kidney or bladder and we are not justified in assuming that because the patient has hematuria and has hypertension that the hypertension makes the bleeding. Therefore, patients who belong in this category should have the benefit of a complete urological study.

Case III. — Hematuria due to Purpura

The third patient was a female, thirty-three years of age, who gave a history of having had a sudden attack of complete urinary retention. This attack lasted four days. During the attack she passed bloody urine and there was some burning on urination associated with a severe backache. The physical examination was negative. Roentgen ray examination and pyelograms were negative. Cystoscopic examination showed many submucous hemorrhages. Search for foci of infection was negative. A tentative diagnosis of hematuria due to purpura was made and the patient referred to the medical service. Further examination verified our diagnosis of purpura and the patient was treated on the medical service.

Discussion: Here then, we are dealing with a patient in whom the bleeding was due to systemic disease, to a condition clearly outside the urinary tract. This again illustrates the importance of looking at the patient as a whole. Treating a patient of this type emperically is, of course, a mistake.

Case IV. — Hematuria due to Kidney Tumor

The fourth patient, a man of forty, complained of painless hematuria only once, and this occurred three days before he came to the hospital. He passed a large quantity of dark bloody urine with clots. The physical examination was negative except for a large kidney tumor on the right side. No blood was found in his urine due to the fact that he had stopped bleeding. A set of pyelograms were made and the pyelogram on the right side showed a filling defect; the pyelogram on the left side was normal. A diagnosis of kidney tumor was made. I operated on the patient and the diagnosis of kidney tumor was verified.

Discussion: Let us reflect for a moment the situation in this patient. The patient was just forty years of age. This was his first and only attack of hematuria. The patient had no pain. There was no loss of weight. The patient, as well as the physician, could see no reason for a complete urological study because of the following facts: It was assumed because of the age of the patient that he had no malignant disease. The fact that he had no pain was repeatedly stressed by the patient and his physician. There was no loss of weight.

Unfortunately, in many cases of hematuria due to malignant disease in the urinary tract, the bleeding stops, the patient has no complaints, and he can see no justification for undergoing a complete study. As you all so well know, the one most important factor in the treatment of malignant disease, be it in the urinary tract or elsewhere, is early diagnosis. It is to be remembered in the treatment of kidney tumors that, when the diagnosis is easy, the operation is very difficult and the prognosis correspondingly bad. If we wish to render the best possible medical care to this group of patients, the diagnosis must be made early.

General Discussion

When the patient presents himself with a history of hematuria, what is the best possible course to pursue?

1. I think that the most important advice that the physician can give to the patient is to call his attention to the fact that blood in the urine always means the presence of serious organic disease and that every patient with hematuria should have the benefit of a complete and careful urological survey. I think it is the duty of every physician, not only to call the attention of the patient to the fact that he has a serious condition, but the physician must demand a complete study.

2. Sometimes it is necessary to repeat the study in that group of cases in which, after the first examination has been made, there may remain in the mind of the physician some doubt. I personally never hesitate to repeat the entire examination.

3. One must also bear in mind the possibility that a patient may have a small lesion which cannot be demonstrated with our present diagnostic armamentarium. This does not relieve the physician of his responsibility to the patient which, in effect, should be to direct the patient's attention to the fact that he may have a small lesion and that it is necessary to repeat the examination. I would like to illustrate, for example, the following situation in which a patient has a small lesion located in one of the renal papilli, which lesion may erode the papillary artery, and in whom the physical examination and the pyelograms may be normal. Bacteriological studies for tuberculosis by means of smears may be negative. Cultures for the tubercle bacillus and guinea pig innoculations should be made in all cases and, as you all so well know, reports cannot be had for six or eight weeks. Often during this period of waiting, the patient may feel that the physician is an alarmist and fails to come back for check-up.

4. The History. I cannot refrain from emphasizing the importance of a careful history which in many cases gives a clue to the possible cause of the bleeding. In my opinion, the only person to take a history is the physician himself. This, I realize, takes a good deal of time but I do not believe that anybody can elicit a history quite as well as can the physician himself. This does not necessarily mean that one must obtain a long history which stresses

many inconsequential points. A careful history with a careful physical examination and blood count often are of great aid in determining what the next steps in the examination must be.

I always ask the patient who comes to me with blood in the urine whether or not the urine is well mixed or not. If the urine is well mixed, the chances are that the hematuria is renal in origin. On the other hand, a two glass test is most informative. If the first urine passed is clear and if the second glass is bloody, the bleeding is probably vesical and the cause for the bleeding will probably be found at the bladder neck. The patient with hematuria who gives you a history of intermittent interruption of his stream probably has a papilloma, a pedunculated middle lobe, or a stone that falls in the neck of his bladder.

The history of recent acute infections, such as head colds, recurring attacks of tonsillitis, the history of infected teeth, are all most important points. As a matter of fact, this is routine on my service at the Presbyterian Hospital. The history of recurring or recent attacks of upper respiratory infections, especially in children, is most informative. If a patient comes in with hematuria and, upon interrogation, states that he has had a bad cold for six months which he cannot throw off, it probably would arouse your suspicions of pulmonary tuberculosis. A patient who gives a history of pleurisy, with or without effusion, or who gives a history of having been in a sanitarium, or who has had an attack of tuberculous tendosynovitis, will at once arouse your suspicions of renal tuberculosis.

The passage of a stone will direct your attention to the fact that the patient may have a stone in the kidney or bladder.

It is scarcely necessary for me to call your attention to the fact that a patient with hematuria due to polycystic disease may direct your attention to the cause of the hematuria by a history of other members of his family having had polycystic disease. Therefore, if a patient states that his father or mother died of polycystic disease, your suspicions must at once be directed to the fact that you may be dealing with a patient who has polycystic disease of the kidney. It is true that in a certain number of patients with polycystic disease there is no family history. On the other hand, it is surprising how frequently one does elicit a history of polycystic disease. As a matter of fact, I ask them about all their aunts, uncles, cousins, second cousins, grandfathers, and grandmothers. I recall a patient with polycystic disease who was only thirty-three years old who consulted me. Upon asking him what his trouble was he said he had a polycystic kidney and that every member of his family who had it died early in life.

The physical examination is most important. Although in a good many instances, the physical examination may be insignificant, on the other hand, in the cases which I just discussed with you, the physical examination was most informative. The rectal examination, in men as well as the vaginal examination in women should never be overlooked. Patients with carcinoma of the prostate and benign prostatic hypertrophy may bleed. Bleeding in a certain number of women is due to carcinoma of the uterus which has ulcerated into the bladder. In some cases of carcinoma of the rectum, the primary cause of bleeding is to be found in the rectum or colon.

Need I call your attention to the fact that hematuria in women deserves one important consideration. One must be sure that the bleeding comes from the bladder by means of a catheter. One must be sure that the patient is not menstruating. This may appear to be trite but I remember several instances in which I was asked to do cystoscopic examinations of women patients because they had hematuria: the presence of hematuria was due to the presence of menstrual bleeding.

After this simple procedure you are ready to subject the patient to laboratory examination, cystoscopic examination, ureteral catheterization, bacteriological examination of urine, etc. Unfortunately, some are prone to start with the laboratory examination first. This is very unfortunate for two reasons. First, because some of the laboratory procedures carried out may not be indicated and second, the laboratory man who makes the report may be wrong and if he gives you the wrong diagnosis, you are off on the wrong foot.

It is perfectly obvious to you that the treatment of hematuria depends upon the cause of the hematuria and that there is no specific in the way of treatment. Unfortunately, I see many patients late in the course of their disease in whom nothing can be done in the way of surgical treatment who give a history of hematuria that is extended over many years and who have had hormones, vaccines, serums, and vitamins. It is interesting to look over the long list of drugs that have been recommended for the treatment of blood in the urine. This is short-sighted therapy and the sooner we disabuse our minds of the fact that when a patient bleeds we ought to give him some sort of medicine, the sooner we will give better care to our patients.

(SLIDES)

(Slide) I would like to call your attention to the fact that the various procedures used are not always without error. We have here a set of intravenous pyelograms made on a patient who had blood in the urine. The left pyelogram is normal; the right pyelogram shows a filling defect compatible with tumor. The pyelograms were repeated. Again, the left pyelogram was normal and the right showed a filling defect. After due deliberation and consultation by all concerned, it was the concensus of opinion that this patient should be operated upon. I operated upon him and found a normal kidney; naturally I did not remove the kidney. The filling defect, in all probability, was due to blood clot.

(Slide) This patient had hematuria, a palpable kidney tumor, and pain in his kidney. The two sets of pyelograms were made: intravenous and retrograde. A diagnosis of kidney tumor was made and I operated upon the patient. No tumor was found.

(Slide) These pyelograms were made in a young man who attended a football game on a very cold day, became chilled, and was seized with pain in the renal area and passed bloody urine. A tentative diagnosis of tumor was made and immediate operation was advised. The patient subsequently consulted me. A second set of pyelograms were made which did not show the filling defect shown on the first set and it was assumed that the filling defect was due to a blood clot which was subsequently passed. The patient returned to his home in Brooklyn, New York, where, on the basis of the first pyelograms, a nephrectomy was done. The reports of the sections showed a marked nephritis.

I cite these few interesting cases to show you how easy one may be led astray by laboratory evidence unless it is checked and rechecked and unless one interprets the findings with a great deal of care and deliberation.

DIAGNOSTIC PEDIATRIC CLINIC

RHEUMATIC FEVER

By HUGH L. DWYER, M. D.
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Twenty years ago it was not an uncommon practice for the physician to attend a child moderately ill with painful, swollen joints and when the symptoms subsided, to dismiss the case without realizing what the future had in store for his patient. He usually examined the heart carefully because he knew that endocarditis frequently accompanied rheumatic joint disease. He prescribed salicylates in large doses and the joint symptoms terminated in a week or two. If there was no heart murmur it was assumed that the child escaped carditis and after admonitions on the need for tonsillectomy, treatment and observation terminated. We did not appreciate the possibility of continuing activity in the disease process and the development of cardiac damage months after the episode of fever and polyarthritis.

At that time streptococcal infection was suspected to be the cause of rheumatic disease and the focal infection theory dominated the scene. It was but natural, therefore, that rheumatic sufferers were subjected to tonsillectomy in the unsubstantiated belief that by this procedure the cause of cardiac infection would be eliminated.

Since that time in a half dozen clinics throughout the country, large groups of rheumatic children have been followed for long periods, ten to twenty years, and we have learned something of the natural history of the disease. It is now generally appreciated that rheumatic fever is a protracted disease and that a positive diagnosis of the rheumatic state implies that all structures of the heart may be, and indeed are likely to be, involved. In several groups of 1,000 or more rheumatic children under observation for ten years, we learn that approximately one-fourth succumbed to the disease. Over a twenty year period nearly one-third of the rheumatic subjects fail to survive. This high death rate as well as the cardiac disorders affecting a good portion of the survivors points to the need for long time convalescent care and the same quality of medical and nursing attention required for the child with tuberculosis or diabetes.

We are slowly coming to the realization that the physician, individually, cannot cope with the problem and that community facilities must be provided for case finding and follow-up services, hospital beds for long time care, and a program of health education to minimize the ravages of rheumatic heart disease.

Rheumatic fever is a major public health problem ranking with tuberculosis and syphilis. Its incidence in the population cannot be determined but from intensive surveys in some areas it is conservatively estimated that one million persons in this country have rheumatic heart disease. According to Martin the incidence rate of rheumatic heart lesions in children is about 0.7 to 1.0 per cent of the school population. Between the ages of 5 and 14 years it is the leading cause of death and even with poor reporting and inaccurate figures for rheumatic fever we are told that it destroys seven times as many children as does poliomyelitis. It is responsible for 90 per cent of all heart disease in persons under 25 years.

CAUSE

The cause of rheumatic fever is still unknown although we believe attacks are precipitated by upper respiratory infections due to Group A hemolytic streptococci. The usual history in a child with an acute infection, or a recurrence of activity, is that of an attack of streptococcal pharyngitis, mild or severe, and after a latent period, usually 1 to 3 weeks, rheumatic manifestations develop. Outbreaks of the disease have been observed following epidemics of pharyngitis in boarding schools and in military posts where large numbers of susceptible individuals are crowded together. It is an urban disease and like tuberculosis, social and economic factors appear to influence the occurrence of rheumatic fever. Unhygienic living conditions, notably dampness, and poor nutrition are noteworthy contributing factors.

The disease is more common in the winter months, November to May, and occurs more frequently in northern climates. Its incidence varies greatly in different localities being more common on the northern Atlantic seaboard and in the Rocky Mountain states.

SYMPTOMS

We will discuss the principal features of rheumatic fever as it occurs in childhood, where its course is marked by alternating periods of exacerbation and freedom from symptoms. The peak of incidence of first attacks is between five and seven years of age; after puberty the frequency of first attacks and recurrences falls off abruptly. In approximately half the cases a family history can be obtained. Its tendency to concentrate in certain families does not mean that the disease is spread by contact. As our knowledge of rheumatic fever increases we may find that individual susceptibility is the determining factor in the development of the disease. Recent studies by May G. Wilson and associates on families of rheumatic clinic patients in New York City present further evidence that susceptibility to rheumatic fever is hereditary. Her studies indicate that the most important factor in the pathogenesis of the disease is the genetic susceptibility of the host.

The variable manifestations of rheumatic fever often make the diagnosis difficult, indeed if not impossible at times. Polyarthritis is probably the most common single manifestation. Carditis is likewise a very common symptom when the disease begins in childhood but usually occurs in conjunction with other manifestations. Other clinical signs frequently observed are fever, chorea, spontaneous nose-bleed, pallor, anemia, erythema and precordial, abdominal and pleural pains.

Fever is one of the most frequent symptoms. There is a tendency toward daily fluctuations of 100° to 101°; or days may pass between these fluctuations. After the subsidence of other clinical signs a low-grade fever may persist for many months as the only sign of activity. Careful recording of the daily rectal temperatures in suspicious cases as well as in convalescents is very important.

The heart is involved in nearly, if not all attacks; if it should escape the first, it seldom does so the second or third time. Once the heart shows clinical signs of involvement the lesions extend with each recurrence in the majority of patients. However, complete functional recovery may take place when infection dies out, even in the presence of valvular disease. When these patients are followed over a ten-year period, three out of five will have developed permanent heart damage.

Early signs of heart disease are tachycardia out of proportion to the fever and a systolic murmur at the apex. This mitral regurgitant murmur may be the result of myocarditis; the heart weakens and dilates, which renders the valve incompetent. In the early stage of rheumatic fever there may be no demonstrable enlargement or valvular disease. These may come within days or perhaps weeks, sometimes not until several exacerbations of rheumatic activity. A proper appraisal often cannot be made of heart damage until many months after an attack. Fulminating attacks of rheumatic fever may quickly involve the heart with pericarditis, valvular disease and enlargement. Stanley Gibson of Chicago found that 44, or 30 per cent of 146 children with rheumatic fever died in the first attack, most of them within a year of the onset of their disease.

In rheumatic valvular disease the mitral valve is most frequently involved, with vegetations on the valve leaflet; later, scarring and valvular deformities occur. Stenosis of the mitral and aortic valves is slow in developing and is not often seen in very young children. The heart may appear to escape injury, an early systolic murmur may disappear for a time and a year or two later a well developed mitral stenosis may be discovered.

Precordial pain comes from pericarditis, and this pain with the friction rub may be of short duration in the early or acute stage of the disease.

The electrocardiogram reveals certain abnormalities that may aid in the diagnosis. Evidence of a disturbance of conduction, such as prolongation of the P-R interval, is the most reliable sign. This procedure is of greatest usefulness in those patients presumably in the quiescent stage to determine whether the activity has entirely disappeared. The same is true of the determination of the sedimentation rate of red blood cells, although both of these laboratory tests may be helpful in those cases that develop insidiously with no other clinical signs than pallor, loss of weight or low-grade fever.

Fleeting pains in the muscles or tendons may mark the onset of the disease but all leg pains are not manifestations of rheumatic fever. Many children have been subjected to tonsillectomy or put to bed for long periods because of so-called "growing" pains, when they are not in danger of rheumatic heart disease. These "growing" pains come on at night after the child falls asleep. They are often severe enough to keep the child awake and crying for an hour or more and may be relieved by heat and massage. These are vague pains in the calf or the thigh, never in the upper extremities nor in joints. They disappear upon awakening in the morning.

Rheumatic leg pains occur in both upper and lower extremities, in the joints, and mostly during the day. They are likely to diminish or disappear when the child is warm in bed during sleep. Oftentimes they are first discovered when the child gets out of bed in the morning. The joint is painful and stiff and this increases on motion causing the child to limp. The patient with rheumatic joint pains will be able to definitely locate the pain and will probably have a history of former attacks or other signs of the disease such as fever, nose-bleed, leucocytosis and increased sedimentation rate.

The diagnosis is not difficult when it is seen in the acute stage with fever and migrating joint pains, or mitral regurgitation. It should be emphasized that severe carditis may develop without any classic signs of rheumatism. We should not think of rheumatic fever in children in terms of joint disease. Polyarthritis is not a constant feature of the disease; it is not nearly as frequent in childhood as it is later in life. When it occurs, the ankles, wrists and knees are most frequently involved. There may be slight swelling but the joints are rarely red, hot and enlarged as occurs in adults. This symptom may last less than a week and the temperature is seldom very high. Occasionally the joint pains may be so mild that the child will limp about but never go to bed. Such patients often have a slight swelling of various joints which is overlooked by the parents.

The skin rash of rheumatic fever usually occurs when polyarthritis is present and the characteristic lesion is erythema marginatum. It appears over the chest and abdomen as a circular, serpiginous lesion with a pale center and a threadlike, slightly elevated, reddened border. Spontaneous severe nose-bleeds and abdominal pain without nausea are frequent and valuable diagnostic symptoms of active rheumatic disease. Coburn found abdominal pain preceded recrudescences more than any other symptom except nose-bleed.

Subcutaneous nodules are pathognomonic of rheumatic fever although the absence of nodules does not mean that the disease does not exist. These nodules are oval, transparent, fibrous bodies that have been compared to tapioca grains, although they may be as small as a pin head. They are superficial and better felt than seen. They are found over the bony prominences on the hands, elbows, ankles, spine, scapula and in the scalp. They may appear suddenly and disappear in a few days or persist for months. Rheumatic nodules, therefore, are not reliable as a diagnostic sign. Doctors Hayes and Gibson of Chicago have recently contributed to our knowledge of the significance of subcutaneous rheumatic nodules. In a series of 167 patients exhibiting nodules they were present on the elbows in 117. Definite signs of heart disease were present in 163 of the 167 patients, and other rheumatic phenomena in every case. Thirty-one per cent of the cases died. These investigators conclude that their continued presence, particularly when new nodules are appearing from time to time, indicate that the rheumatic process is active and the mortality rate of patients with nodules is so high that they are of great prognostic import.

The earlier the age of onset the greater is the chance of developing cardiac failure with an unfavorable outcome before adolescence. Enlargement of the heart, determined best by X-ray examination in several positions, also portends an unfavorable outcome.

There is no positive laboratory test for rheumatic fever. Leucocytosis usually accompanies active infection and frequently persists after clinical evidence has disappeared. In the acute phase this ranges from 12,000 to 16,000. A persistent leucocytosis of 10,000 or more in a rheumatic subject following clinical manifestation is evidence of activity.

The sedimentation of red blood cells is less subject to fluctuation than the leucocyte count. It is a simple test now in wide use in the physician's office. It is doubtless the most valuable single blood study in rheumatic fever and is especially useful in judging the activity of the disease during convalescence.

Treatment

The management of rheumatic fever in its active phase consists of symptomatic relief from pain and fever with salicylates, the protection of the child against new infections, and strict bed rest. In the acute phase of the disease the patient should be in a hospital under skilled medical and nursing supervision. Except for the subacute cases the patient should be recumbent. This may mean months in bed with a loss of a year or more of school work. It is often difficult for the patient and parents to become reconciled to this regime. Most attacks last three to four months and they may last three to four years. Long bed rest is as necessary in rheumatic fever as it is in tuberculosis. Whether this should be in the child's home or a sanatorium depends much upon the type of home and the community facilities. In any event the patient must be protected against respiratory infections, cold and dampness, and on a dietary regime to promote health and well being.

CASE PRESENTATION

We have here three boys, all rheumatic subjects, to illustrate some of the features of the disease of which we have been speaking. Needless to say, they are in the inactive phase of the disease and are in a special school for handicapped children, the Spaulding School in Chicago. Their activities are restricted but they are getting their education under proper supervision.

CASE 1.

Frank, aged 15 years, entered the Children's Memorial Hospital in 1933 when he was 5 with a systolic murmur. He had had a few bouts with rheumatic fever; his mother has been in the hospital with rheumatic fever and one other child in the family died of the disease.

I note that he had his tonsils removed after he entered the hospital. The majority of students of rheumatic fever contend that there is nothing to be gained by a tonsillectomy; it does not prevent first attacks or recurrences. However there is no unanimity of opinion on this question. Dr. Albert Kaiser of Rochester, New York, after a large experience in evaluating tonsillectomy and its effect on health, believes that it does lessen the likelihood of recurrent attacks in rheumatic subjects. I am inclined to favor tonsillectomy during the inactive stage of the disease and I note that all three boys before us have had the tonsils out. This should never be done during the active stage and I think it is well to prepare the rheumatic child for the operation by the administration of one of the sulfonamide compounds for three or four days before operating.

Four years after Frank's mitral regurgitant murmur was discovered, the clinical record shows aortic stenosis was found. It took some time to develop this, at least it was not recorded until four years after his first hospital admission. In 1941 he had a recurrence of rheumatic fever; therefore his disease has been inactive only a short time and he is not out of danger of new attacks.

However he is in a special school for handicapped children. His activities are supervised but there is no disposition to make him an invalid. At school he does not run upstairs, he uses ramps. He rests several times each day. In the summer he attends a supervised camp for "cardiac" children. His mental tests show a high rating and he is getting his education in spite of his cardiac infirmity.

CASE 2.

The next boy, Edwin, is 15 years of age. He came to the hospital in 1933 with a diagnosis of "heart disease," when he was 5. All three of these boys had carditis early, and it is noteworthy that this may be the only symptom when the disease becomes manifest at 4 or 5 years of age. His grandmother had "rheumatism." He was not allowed to remain in the hospital but was kept in bed at home for six months. This was all right if he were actually kept recumbent and protected from his family and his friends with respiratory infections.

Four years after his first illness his tonsils were removed (May, 1937). The following two years he got along very well in school but in 1939 he became acutely ill and his physician sent him to the Children's Memorial Hospital with a tentative diagnosis of appendicitis. Preceding this abdominal attack he had frequent spontaneous nose bleeds (three times a week) which was evidence of a recrudescence of active infection. At the Children's Hospital it was found that he had the abdominal manifestations of rheumatic fever and he escaped an abdominal operation.

The hospital chart shows that he had subcutaneous nodules and skin lesions.

Edwin, did you ever see your nodules?

PATIENT: Yes.

DR. DWYER: How many did you have?

PATIENT: I had about sixty that I could count.

DR. DWYER: He had about sixty—probably on his elbows, wrist, ankles and scalp—which are usually better felt than seen.

Did you have any rash on you?

PATIENT: Yes, erythema.

DR. DWYER: I didn't coach him but I note on his chart he had annular erythema. I suppose this disappeared quickly as nodules and erythema lesions are likely to be of short duration. All this occurred in 1940 and now has mitral and aortic stenosis with a thrill in the suprasternal notch. This is an intelligent child and like Frank has an I. Q. rating of 111. He is in the Spaulding School and he is being taught how to live with his cardiac disability.

Do you take rest periods at school?

PATIENT: Yes, one a day.

DR. DWYER: Do you ever run upstairs?

PATIENT: No.

DR. DWYER: Do you play games, basketball or anything?

PATIENT: Yes.

DR. DWYER: No doubt he has supervised play and does not engage in competitive sports. The institution does not make invalids out of these boys but teaches them to lead normal lives.

CASE 3.

The third patient is Chester, 11 years of age. He entered St. Luke's Hospital when he was seven with a "loud systolic murmur at the apex." I note that he had a pulse rate of 120 to 130 with a temperature of 97.4° F., a tachycardia without fever. He remained at the hospital six months. In 1942 he had a recurrence and he was kept recumbent for three months while he had an increased sedimentation rate. When this returned to normal he was allowed bathroom privileges for two months, then for another two months he was allowed to be up and about only two hours each afternoon.

He returned to school in October, 1943, after he had missed several years. He is still young and he must be carefully watched for any sign of activity. A throat infection would be particularly hazardous to this child. He now has mitral stenosis.

Each of these patients had a recurrent attack of the disease after a tonsillectomy. None of them had very much medication. For a short period one had digitalis for heart failure.

Time will not permit a discussion of congestive heart failure which is characterized by edema beginning in the face, and enlargement of the liver due to increased venous pressure. Suffice it to say that digitalis to lower the heart rate and combat edema is not without danger and often disappointing. The possibility of adding digitalis toxicity to the overburdened patient is always present. It is safer to treat edema with one of the xanthin diuretics, theocalcin or theodate in 1 gm. doses three times a day.

Those who have given sulfanilamide in the active stage of rheumatic fever warn us that it regularly causes toxic symptoms and should not be used.

INACTIVE PHASE

After several weeks or months in bed, when all signs of infection have disappeared and salicylates have not been used for two or three weeks, the question of relaxing the regime of strict bed rest will arise. The child should not be kept in bed too long. The normal pulse rate, recorded while the child is asleep, the freedom from fever as determined by twice a day rectal temperature readings and the disappearance of all clinical signs of activity should be checked with the sedimentation rate and the leucocyte count. If there is no evidence of active infection the child may be allowed to feed himself and later, to sit in a chair for a half hour in the afternoon. When this much activity is followed with no significant increase in the pulse rate, bathroom privileges and longer periods out of bed are permitted. There can be no hard and fast rule governing the child's activities. If the sedimentation rate has been increased during the disease and has returned to normal, it is a good indication that infection has subsided.

Secondary anemia tends to come on in active rheumatic infection and to disappear when activity subsides. A rise in hemoglobin values may help to determine the cessation of active infection.

PROPHYLAXIS

Sulfanilamide and other sulfonamides are being used experimentally as a prophylactic measure against recurring attacks of rheumatic fever during the inactive phase in rheumatic children. The drug is usually started in October or November and continued through May. Coburn and Moore gave sulfanilamide, in daily doses of 2 to 3 gm. depending upon weight, to 184 rheumatic subjects throughout the school year, October to July. The incidence of rheumatic fever attacks in this group was 1 per cent, compared to an expectancy rate of 35 per cent.

Thomas and her associates at the Johns Hopkins Hospital gave sulfanilamide to 55 persons with a recent history of rheumatic fever from November through June; to some, for four years. In amounts of 20 grains (1.2 gm.) daily, divided into two doses, 12 hours apart, most of the patients took the drug without the slightest discomfort. The authors state that the "toxic symptoms which follow larger therapeutic doses were rarely if ever observed." While taking sulfanilamide, none of the 55 subjects had a major attack of rheumatic fever. In the control group of 67 rheumatic subjects not taking the drug, fifteen major attacks of rheumatic fever occurred during this four year period and three died.

Not all reports on the use of sulfanilamide in the prevention of rheumatic recrudescences are so favorable. Recent reports of several deaths from its use in this manner remind us that the prevention of rheumatic attacks is still a research problem.

DIAGNOSTIC SURGICAL CLINIC

Appendicitis and Its Complications in Infancy and Childhood

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Discussing the subject of appendicitis and its complications in infancy and childhood may seem to some like going too far afield in the plan of specialization and wonder why we should restrict it to children. There is much to be said in behalf of this statement. Those of you who have read Ladd's introduction to his book on Abdominal Surgery in Infancy and Childhood know that he states that a child is not a diminutive person, that it is not the full-grown human organism that we see in the adult. I should like to add to that remark and state that while a child is full term at birth, it is still premature as far as physical protection, social relationships, mentality, power to interpret surroundings and sensations are concerned. So we are dealing, then, at the start with a premature condition in many respects, and only those who are familiar with children can understand this.

Why is appendicitis different in infancy and childhood than in adult life? That is a statement that you have heard many times and I think most of you know the answer. Knowing the answer and putting it into active, successful operation is another thing. If we want to skip the art of approach to a child and go directly to the cause without recognizing the fact that we are dealing with a premature individual, that requires art, tact and diplomacy, we are ignoring a very important principle in the successful management of children. When we recognize this factor we have gotten over a big barrier in enlisting the child's cooperation and in arriving at a correct diagnosis.

We know that a child's sensibility is less acute than an adult's. His power of interpretation as to where his pain has been and is, is different. You will notice frequently that when a child is very ill and you ask him, "Sonny, how do you feel?" his answer is "Fine." That is the answer we get most of the time in our work in the children's ward at the Cook County Hospital in Chicago, and if we accepted that as final we would be misled most of the time. We must constantly keep in mind that in acute appendicitis a child places its hand on the umbilical region rather than over the right lower quadrant when it is asked the location of the pain. Also the leucocyte count is much higher than one sees in an adult.

I should like to talk to you for a little while about our cases that cover a period of probably seven or eight years. Maybe it is the duty of some of us who have the privilege of working with children in surgery to present to you from time to time our experiences. That is what I want to do this afternoon.

In our work at the Cook County Hospital at the present time we are following up a program that was developed five or six years ago by Dr. Miller and his associates with reference to the treatment of acute appendicitis in children. In the last three years we have followed his method of treatment and his classification or division of appendicitis. We have continued the work and have gathered further proof of the value of this classification.

I do not want to spend time on the diagnosis of acute appendicitis. I want to mention that we have three groups. Group I consists of the average case of acute appendicitis with more or less the typical history of acute appendicitis: the child is sick probably eighteen hours, twenty-four hours, may be thirty-six hours, and represents a group that we consider should be operated upon immediately. It covers the average run of acute appendicitis that we operate day or night and is the case of acute simple appendicitis. The findings usually consist of some pain and tenderness in the right lower quadrant and there may or may not be rigidity. We do not feel that we should wait for rigidity to develop after other findings are quite conclusive that we are dealing with acute simple appendicitis. Frequently when I am called in consultation I am told there is as yet no rigidity. Why should we wait until rigidity develops? That is a very poor philosophy; it is dangerous. When rigidity develops we are ushered into the first phase of the complications of acute appendicitis. Complications we all want to avoid and so we operate immediately and it is in this group that we all boast of a low mortality rate. In Dr. Miller's series 629 patients and in our series 441 patients belonged to Group I whose mortality was zero. That is nothing to be boastful about; I am sure all of you surgeons have similar experiences.

Our second group constitutes a group of patients who have been sick probably thirty-six hours and longer, maybe seventy-two hours, who come in not acutely ill. They may walk slightly stooped because of pain in the right lower quadrant. In the few days preceding there have been symptoms of acute appendicitis. We examine the patient and we find this patient has a palpable mass in the right lower quadrant or in that vicinity. In our opinion that represents the first complication of appendicitis. I am not speaking now about postoperative complications of appendicitis; I am speaking about complications of acute appendicitis. When we have this palpable mass with rigidity and pain, we are dealing with a complication that represents either a diffuse phlegmonous reaction in the wall of the appendix to which there have been summoned omentum and probably loops of intestine to wall it off without actual perforation, or it may have actually perforated, producing a small abscess and likewise a palpable mass.

How are you going to distinguish between those two conditions? You probably have all heard the late Dr. Charles Mayo say, "The greatest problem of abdominal diagnosis is the abdominal wall." We cannot tell whether we are dealing with a phlegmonous inflammation to which the omentum has become adherent by plastic exudation or whether we are dealing with an actual leak, a perforation which represents suppuration and abscess formation. We cannot tell the difference at the time. We sometimes think that by the temperature, by the leukocyte count, by the size of the mass, by the amount of tenderness and rigidity we can distinguish between a phlegmonous inflammation and an abscess remaining localized. Probably within a week, maybe less time than that, we can tell. Our experience has been that if we are dealing with a phlegmonous inflammation only, the mass, on conservative management, disappears much more rapidly, probably in six or eight days, whereas if we are dealing with an abscess, a suppurative lesion, it takes longer for resolution to take place, may be two weeks or three weeks before the mass disappears. Differentiation is made very largely on the basis of time and we have not been wrong in many instances.

The palpable mass felt generally is in the right lower quadrant or near the midline. Sometimes it is felt directly in the pelvis. This group we treat conservatively, usually nothing by mouth for a few days; intravenous glucose; sulfonamides; sometimes plasma; sometimes heat; sometimes cold packs to the abdomen. I do not know just how much good the packs do—I am open minded on this. After a few days these children are fed again and returned to a pretty normal existence, but they are kept in bed until the mass disappears; then they are sent home, to return probably in three months for an interval appendectomy.

In Dr. Miller's series there were 203 patients in Group II and the mortality in that group was 2.5 per cent, while in our series there were 74 with a mortality of 2.7 per cent. We felt at that time that if we could treat these children with localized abscesses, in other words, localized peritonitis, conservatively and have them return two, three, four months later for an interval appendectomy, and bring that mortality from 8 per cent with immediate surgery down to 2.5 per cent with delayed surgery that such a plan was worth trying.

In our series of 74, in Group II, our mortality has remained about the same, which in our opinion is a justification for continuing this type of management.

The third group consists of those patients who have been sick probably three to five days or even a week. Our time measure of illness is not very dependable in children because a child will play for a day without complaining to his mother about pain. The child comes to the hospital and the mother may state that the child has been sick for one or maybe several days and it already has a generalized peritonitis. Another child may come in four or five days after the onset or known onset of the illness with a generalized peritonitis also.

In this Group III we are dealing with a ruptured appendix that has not been walled off by the omentum and consequently has disseminated throughout the peritoneal cavity and resulted in a generalized peritonitis. This group really constitutes the seriously ill group in acute appendicitis. It represents the *major* of major complications, in my opinion; it represents a group that we have not been able to change the mortality in very much in all these many years. It is still the same group in which the old surgeons long before appendectomies were done had their mortalities, not in those simple acute cases, nor in the group that form palpable masses. The group in which we are having our mortalities is the same group in which they had their mortalities, so we still have the same problem to deal with.

In Dr. Miller's series there were 281 with generalized peritonitis that were operated upon immediately with a mortality rate of 12.8 per cent and 25 not operated upon with a mortality of 80 per cent. This constitutes an emergency surgical condition if the patient is in condition for operation. One of the greatest contributions in the reduction of surgical mortality throughout the whole field of surgery has been the recognition of the fact that one must prepare his patient for surgery regardless of how ill he may be. If the patient is in shock, naturally one would not operate. If the patient is on the verge of shock one is going to resuscitate the patient with all the measures of resusciation that we know of before one operates. So with our Group III, appendicitis with perforation and generalized peritonitis, surgery is immediately undertaken.

In Miller's group III the mortality was 12+ per cent. In our series of group III of a later date we have reduced that to 7 per cent. That makes a very low mortality rate. In our ward, the children's surgical ward of the County Hospital, we are not ashamed of these figures, 7.4 per cent in generalized peritonitis with immediate surgery, after, of course, we have used all the means of restoration such as infusions of blood, plasma, glucose, and the administration of sedatives and sulfonamides.

In the group of generalized peritonitis not operated upon the mortality was too high even to think of following that treatment. We had some fatalities, naturally, among patients who came in in stages of inoperability or in the late stages of shock, with definite generalized peritonitis. These patients have a poor chance, but where they have a chance they do not all die if they are not operated upon. There was a time in the Cook County Hospital when there was a policy established of "hands off" the patient who had been sick longer than sixty hours with acute appendicitis. Why is it that we have changed? It has not been because the surgeons have gotten appreciably better in their technical ability. It is because we have recognized the tremendous importance of preoperative preparation and postoperative management.

Why is it that the mortality in our series should be less than the mortality in Dr. Miller's series? It is nothing that we have done, nor has it been anything particularly that he has wilfully neglected. His concept and division of appendicitis into these three groups has met with quite universal acceptance on the staff of the Cook County Hospital. The series that I am reporting to you this afternoon were operated upon by fifty-two different surgeons and the sum total of the mortality of all three groups is 2.2 per cent. The all-inclusive mortality in Miller's group was 5.4 per cent. Why is that? It is not because we have better surgeons. When we can present to you a group of almost 700 cases of acute appendicitis operated upon by fifty-two different surgeons, what does that reflect? First of all, it reflects that there is being established in our institution at the Cook County Hospital a very definite consciousness of these three groups of appendicitis and their management. It has furthermore been established that those who operate are well trained.

Another thing that they have learned, because we have hammered away at it, because Dr. Ladd has hammered away at it, is that when you are operating on children you are dealing with tissues that are delicate and tender. That is something we should like to drive home. It is not that we want to plead that you limit your surgery to pediatric surgery at all; it is to enable you to appreciate what it means to operate on a child as compared to an adult.

Our work takes us into newborn infants twenty-four hours old. We cannot quite go at that as one would at a gallbladder in a person who weighs 200 pounds. It is the development of an appreciation of what it means, and our staff at Cook County has developed that appreciation. Our surgeons for the most part when called upon to operate a child a year or two years old, realize that the tissues must be handled gently, delicately, carefully.

Another factor accounting for this reduction in mortality has been the fact that when Dr. Miller's series first began he was trying to develop a new system. You know how difficult it is to institute a new plan and how hard it is to get the whole personnel to follow it. In our case it is a difficult and involved thing to get them all to think the way we think. It cannot be done in a month or a year. It takes some years. When our series began we had the benefit of that added training. We have had a more generous access to blood banks and blood transfusions and blood plasma than Dr. Miller. We have been able to employ more universally the sulfonamides than Dr. Miller did in his series. A further reason is that 64 per cent of our series were classified in group I when only 54 per cent of Miller's series were classified in group I. This means that a much larger number of patients are coming in earlier for surgery.

We realize that all do not agree with us and that there is no uniform opinion that appendicitis should be treated as we have outlined. There are some clinics in this country with expertly trained surgeons who have even a lower rate of mortality than we have at the Cook County Hospital. However, I am rather proud to be able to present to you a series of almost 700 cases of acute appendicitis in children in all stages, many of them with peritonitis, or with abdominal masses, operated upon by fifty-two different surgeons, 90 per cent of the surgeons in process of training, such as associates, residents and senior interns, with a mortality of only 2.2 per cent.

DIAGNOSTIC MEDICAL CLINIC

Management of Addison's Disease

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I wish to thank you for the privilege of being here and of speaking to you this afternoon about Addison's disease. I wish to thank Dr. W. O. Thompson particularly for his courtesy in providing three patients with Addison's disease to whom I shall refer from time to time.

It always interests me to reflect that the first case of Addison's disease was recorded by Richard Bright of Bright's disease fame about twenty years before Addison reported his famous cases.

First, I would like to present a few fundamental concepts regarding the adrenal gland that guide us in the treatment of Addison's disease.

1. The adrenals are vital organs. In nearly all animals adrenalectomy leads to death within a few days, but as yet there is no agreement as to what change constitutes the primary cause of death.

2. The vital function is centered in the adrenal cortex and appears to operate principally by delivery of a mixture of substances into the blood stream. Injection of suitable extracts into adrenalectomized animals controls these symptoms and prevents death.

3. Investigation has shown that the activity of cortical extracts is due to a mixture of steroids. Up to July, 1942, twenty-eight steroids had been obtained in crystalline form, and some had been partially synthesized. Many less well understood compounds from the adrenal cortex are being examined. Six or seven compounds possess various activities which may either prolong life in adrenalectomized animals or affect single insufficiency symptoms.

Physiologically, adrenalectomy in animals produces the following important changes: (1) Excretion of Na, Cl, and water. (2) Retention of potassium and urea. (3) Muscle weakness. (4) Abnormal carbohydrate metabolism. Poor corbohydrate absorption + inefficient gluconeogenesis → decreased liver glycogen → hypoglycemia. (5) Reduced resistance to trauma including mechanical shock, hypoglycemia, cold, and toxins.

In clinical adrenal failure these features have their counterpart, producing asthenia, digestive disturbances, tendency to hypoglycemia, and lowered resistance to shock. These as well as the melanoderma usually develop insidiously.

In severe insufficiency serious psychotic symptoms may arise, and with crisis rapid respiration and high fever may suggest the presence of pneumonia and recall the fact that such changes occur in adrenal cortical hemorrhage. The menses are surprisingly little affected, as a rule, and pregnancy is possible. Men with Addison's disease do not lose sexual power and may remain fertile.

Various changes in Addison's disease and in adrenal failure in animals are clinically useful as bases for diagnostic measures, and others are of relatively little value. These include (1) abnormal heart size and abnormal electrocardiograms, (2) abnormal electroencephalograms, (3) abnormal creatine metabolism and reduced hippuric acid excretion, (4) abnormal amino-acid tolerance, (5) abnormal plasma protein levels, and (6) low excretion of 17-ketosteroids.

The level of urinary 17-ketosteroids is of distinct diagnostic value. The two sources of 17-ketosteroids (androgens or masculinizing hormones) are the interstitial cells of the testes and the adrenal cortex. In women the only source is in the cells of the adrenal cortex. Therefore, in men who have Addison's disease the 17-ketosteroid output in the urine is lacking that part which comes from the adrenal cortex. It is, therefore, about halfway between zero and the normal. In women, however, where no source, or practically no source, is left for these materials, its excretion approximates zero.

These three patients demonstrate various degrees of the typical picture of Addison's disease. I cannot demonstrate in detail the various changes. I will speak first about the gentleman on the left. This man has had Addison's disease symptoms since 26 years of age, nine years ago. His symptoms were those of fatigue and weakness. Addison described the syndrome originally as "general languor and debility, feeble heart action, irritable stomach with a dingy or smoky discoloration of the whole surface of the body." The facies of the untreated Addison's patient shows this peculiar muscle weakness. This weakness involves not only the skeletal muscles but also the muscles of the gastrointestinal and cardiovascular systems.

Patients react to the weakness of Addison's disease with quiet resignation. Their faces show no suggestion of anxiety, no glint in the eye, nor activity commonly seen in toxemia or psychoneurosis. If the facial weakness is pronounced, the facies is suggestive of myasthenia gravis.

Because of the lack of tone throughout the cardiovascular system, the blood pressure usually falls, sometimes to very low levels. A postural hypotension exists, so that when the patient stands the systolic pressure falls 20 or 30 points. The blood pressure in Addison's disease, however, may be maintained within normal range, in spite of serious cortical failure and to within a few hours of crisis.

Digestive disturbances in Addison's disease are almost as constant as weakness, which apparently occurs in all cases. Digestive disturbances are present in perhaps 90 per cent of the cases and are reflected at least by anorexia. Episodes of nausea and vomiting may occur from time to time. Vomiting and diarrhea, however, in causing chloride and water loss, are likely to bring on a typical crisis. Crisis has occurred in the cases of Mr. Patterson and Mrs. Ray a great number of times. Mrs. Ray has not had crisis during treatment for the past two years. Prior to that she had Addison's crisis so many times that she is no longer sure of the number. Mr. Patterson, who has been in crisis eighteen times, is a walking advertisement for his physicians and for the methods used in treatment. Addison's crisis is very serious and must be treated very energetically and meticulously.

In Addison's disease constipation is more common than diarrhea, although both occur. Sometimes there are vague abdominal pains. Some cases finally diagnosed Addison's disease have been treated as functional indigestion for years. One of my patients who was treated for indigestion for fourteen years, during which time leukoderma was present, was finally proven to have Addison's disease.

With all of these digestive symptoms it is perhaps surprising that more weight is not lost. Mrs. Ray's weight was as low as 65 pounds, probably because she had a chronic miliary type of tuberculosis. In my own experience the weight loss in Addison's disease approximates 20 or 25 pounds by the time that other symptoms are severe enough to require attention.

The pigmentation, the third outstanding clinical evidence of the disease, is fairly marked in the patient in the middle. The pigment in the average case is perhaps best described again in the original words of Addison, "a smoky or dingy discoloration of the skin of the entire body." The smoky color of the pigment is usually characteristic. It always looks to me as though a good rub with a wash cloth might make the pigment lighter, as the skin often has a dirty appearance. Often the pigment is exaggerated over areas of pressure, over scars, or in creases such as palmar creases. Blotches may be present in the mucous membrane of the cheeks and lips, as in Mr. Patterson's case. Another type of pigmentation is present in Mrs. Ray's case, namely, black freckles. Usually the freckles are small and tend to increase in number and in depth of pigmentation and frequently look like India ink. When leukoderma occurs the nature of the disease may be suggested by other types of pigmentation such as hyperpigmentation of the nipples.

In some cases argyria, hemochromatosis, or arsenical dermatitis may be suspected. Vagabonds' disease, melanotic sarcoma, acanthosis nigricans, or even pigmentary changes during pregnancy may raise a question. Methods available for diagnosis are biopsy and proper staining of the skin, appropriate urine tests, and further observation.

Hypoglycemia is common and may produce serious symptoms. The hypoglycemia tends to occur after ordinary periods of fasting, after exertion, or during extra strain such as infection. Because of lack of resistance to shock, people with Addison's disease resist hypoglycemia poorly.

Certain laboratory tests help to corroborate or refute the diagnosis of Addison's disease. Probably every Addison's patient should have an x-ray of the chest because tuberculosis is a probable cause of Addison's disease, although probably not in more than 50 per cent of the cases. Almost all the rest, excluding a few due to malignancy, are due to a peculiar atrophy of the cortex of the adrenal, the etiology of which is unknown.

The adrenal areas may be x-rayed. Calcification in the adrenal may be helpful clinically, so it is well to know that adrenal calcification may occur in otherwise healthy individuals.

In most instances there is a lack of acid in the stomach. The basal metabolic rate tends to be low, in the neighborhood of 20 per cent below normal.

Certain typical changes in the blood sometimes are hard to interpret accurately because various elements are changed in their apparent levels by the amount of hydration of the blood itself, so that in concentrated blood a low serum sodium might appear to be within the normal range. Urea and potassium which would be normal in normal hydration may appear high when the patient is sufficiently dehydrated. These things must be kept carefully in mind in handling the patient ill with Addison's disease, because unfortunately the plasma volume cannot be measured accurately and quickly.

The blood count is useful. The hematocrit level gives us an indication of the amount of hydration, particularly after repeated determinations. The chlorides are typically low; the sodium is low; and the potassium and urea tend to be high. The carbon dioxide combining power in patients ill with Addison's disease approaches an acidotic level. All of these changes, however, may remain within normal limits in a patient in Addison's crisis.

One particularly good test* has been described in two procedures by Robinson, Power and Kepler for use in the diagnosis of Addison's disease. Procedure No. 1: The water excretion test depends upon the peculiar lag in excretion after the ingestion of large quantities of water. Patients eventually excrete the water but tend to become dehydrated. This lag period can be simply shown by collecting urine during the conditions of the test from 10:30 at night until 7:30 the following morning, and then collecting hourly specimens during the morning after the ingestion of a large amount of water in definitely prescribed quantities. Sometimes the hourly specimens in the morning are so difficult to obtain that an average mean must be calculated. When the test is positive, no single specimen during the morning exceeds that of the night specimen.

Procedure 2 of the test determines the chloride balance and the urea balance with the water excretion. The test is valuable because of its accuracy if properly interpreted, and it can be done wherever blood and urinary chlorides and urea can be measured.

We examined this test more than 200 times, partly to become acquainted with the test and partly for diagnosis of Addison's disease. Among these 200 tests, fifteen patients with untreated Addison's disease had positive tests. Twelve patients who had pituitary disease, extremely low 17-ketosteroid excretion, and other evidence of adrenal failure showed positive tests. Four Addison's patients showed a positive test before treatment and a negative test after treatment.

We do not contend, however, that the test in itself is a good index of the value of treatment.

Certain false positives may be present. Obviously, a test which is based on water excretion, chloride excretion, and urea excretion is difficult to interpret in terms of adrenal cortical failure if other diseases are present which seriously disturb these particular metabolisms. These conditions include intestinal obstruction, renal tuberculosis, uncontrolled diabetes mellitus, diabetes insipidus, cachexia, elevated blood urea, and water retention.

The Cutler, Power and Wilder test is accurate but not practical especially under hospital conditions during the War.

Tests based on the production of severe symptoms or crisis by sodium withdrawal or potassium feeding are associated with a distinct risk. The presence of insulin sensitivity in Addison's disease is well established, but I doubt that its use is ever warranted as a diagnostic procedure.

In the treatment of Addison's disease, we give our residents and students certain specific instructions.

1. Give no transfusion unless the necessity is extreme, as a transfusion reaction may be fatal. If transfusion is imperative do not give whole blood. The least dangerous type is plasma freshly separated from fasting blood.

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* Footnote: "Procedure 1 (based on volume of urine): 'The water test.' On the day before the test the patient eats three ordinary meals but omits extra salt. He is requested not to eat or drink anything after 6 o'clock in the evening. Until this time he may drink water as desired. At 10:30 p. m. he is requested to empty his bladder and discard the urine. All urine which is voided from then on until and including 7:30 a. m. is collected. The volume of this urine is measured and saved for chemical analysis if this should be necessary later. Breakfast is omitted. The patient is asked to void again at 8:30 a. m. and immediately thereafter he is given 20 cc. of water per kilogram of body weight (9 cc. per pound). He is asked to drink this within the next forty-five minutes. At 9:30, 10:30, 11:30 a. m. and 12:30 p. m. he is requested to empty his bladder. In order to eliminate the effects of exercise and posture on urinary excretion, he is kept at rest in bed except when up to void. Each specimen is kept in a separate container. The volume of the largest one of these four specimens is measured.

Under these conditions some patients having Addison's disease will excrete so little urine that they are unable to void more than once or twice during the entire morning. In such instances the amount of urine excreted per hour may be calculated; frequently, however, such calculations are unnecessary because of the very low urinary output throughout the entire morning."

"Procedure 2 (based on chemistry of blood and urine). To carry on with this procedure blood is drawn (preferably under oil) while the patient is still fasting, and the plasma analyzed for its content of urea and chloride. The specimen of urine which was voided during the night is also analyzed for urea and chloride. From these four determinations and from the results obtained from procedure 1 the following equation is solved:

$$A = \frac{\text{Urea in urine (mg. per cent)}}{\text{Urea in plasma (mg. per cent)}} \times \frac{\text{Chloride in plasma (mg. per cent)}}{\text{Chloride in urine (mg. per cent)}} \times \frac{\text{Volume of day urine (c.c.)}}{\text{Volume of night urine (c.c.)}}$$

The term "day urine" applies to the largest of the hourly specimens voided during the day; "night urine," to the entire amount which was voided from 10:30 p. m. to 7:30 a. m. It is immaterial how these values are expressed provided that the same method be used throughout the equation. For example, if the concentration of plasma chloride is expressed as milligrams of sodium chloride per 100 cc., the concentration of urinary chloride should be expressed in the same manner."

2. Do not give a tuberculin test; the patient might develop a fever which will not subside. Without evidence of active tuberculosis, the treatment would not be changed.

3. Do not give insulin. Addison's patients run sufficient risk of hypoglycemia; do not increase it. Coexisting Addison's disease and diabetes mellitus is extremely rare.

4. Do not give thyroid extract unless the indications are unmistakable and then with caution.

5. Do not treat tuberculosis of the adrenal glands with x-ray since a crisis may supervene.

6. Never withhold potassium when desoxycorticosterone treatment is used. Experience has led us to look with grave suspicion upon abnormally low, serum potassium levels.

Types of therapy available for Addison's disease include the following:

1. Dietary.

a. Frequent feedings with high carbohydrate intake are advisable in all cases.

b. Low potassium diet may be useful when sodium chloride alone or sodium chloride plus extract is being used.

c. Sodium chloride may be given orally in solution or as enteric-coated tablets in doses of 5 to 25 Gm. per day.

2. Adrenal extract.

a. The oral preparation of adrenal extract, we believe, is too weak to be of practical value.

b. Extracts for injection (Upjohn, Wilson, and Parke-Davis and Co.) may be used alone. Amounts under 5.0 cc. per day are probably less than adequate in many cases. The cost of this therapy averages $80.00 per month or more, which is an obstacle to continued use for most patients.

3. Desoxycorticosterone acetate.

a. Tablets for sublingual use are effective, but a dose four or five times larger than that used in injection is required.

b. Desoxycorticosterone acetate in propylene glycol is not essentially different from tablets in effect.

c. Desoxycorticosterone acetate in oil, 5.0 mg. per cc., is available (Cortate — Schering; Percorten — Ciba). A dosage of 2.5 mg. per day is commonly effective. The cost per month is approximately $15.

d. Pellets of desoxycorticosterone have not been made generally available. We have used 75 mg. cylindroids prepared by the Schering Corporation and have found that approximately 1.0 mg. per day is absorbed from each 300 mg. implanted. Thorn and others have used pellets weighing 125 mg. made by the Ciba Company.

In Mr. Patterson's case Wilson's extract has been given for a long time in doses of 5 cc. twice a day. He also has been given 12 Gm. of sodium chloride per day and 4 Gm. of sodium bicarbonate in capsules. We give our sodium chloride in enteric-coated tablets.

Mrs. Ray is well maintained on 30 cc. per day of Wilson's cortical extract, an amount which is not generally useful because of cost. In her case 20 cc. per day is considered inadequate treatment.

Mr. Pavay, the gentleman in the center, has had pellets, Schering's 75 mg. cylindroids, implanted subcutaneously, a dose of 450 mg. These pellets last him four to nine months without the use of extra salt. Dr. Thompson tells me that the clinical status of this patient was better on 450 mg. of pellets than on 5.0 mg. of desoxycorticosterone daily by injection. At first thought one might assume that this means the patient would be better with larger injections. I believe the opposite is true. Actual weighing of pellets after many months of implantation has taught us that 1.0 mg. per day is absorbed from four 75 mg. pellets of the Schering type. Most of our patients do best on small doses amounting to between 1.0 mg. and 3.0 mg. per day. Larger doses may not only be useless but harmful.

Extract probably represents the best replacement of adrenal cortex hormone, if it is available in adequate amounts, but the extract is expensive, it is bulky, the dose must be continuous and large, and the injections themselves are a disadvantage.

Desoxycorticosterone is cheaper and more potent; injections need to be given less frequently and are small. If pellets can be implanted, no injections are needed for a period of perhaps eight months to a year or more.

When other hormones of the adrenal cortex are synthesized, treatment will be greatly improved over that now available with desoxycorticosterone. I think that it has not been emphasized sufficiently that desoxycorticosterone only influences the salt and water metabolism and does not protect the patient against infection, hypoglycemia, or shock.

Treatment of the Average Case

Our handling of an uncomplicated case of Addison's disease may be outlined as follows. The patient is hospitalized and, if not ill, is allowed to be about the room. Six Gm. of sodium chloride as enteric-coated tablets and 5 mg. of desoxycorticosterone in oil are given daily. Treatment should not be hurried; as better physiologic result may be expected if sufficient time is allowed. Frequently, the patient who has been ill for many months does not show much response to treatment for a number of weeks.

The diet is not restricted but should be calculated so that a record of the caloric intake may be kept. The diet is high in carbohydrates. Nourishment is given between meals and at bedtime, and if any tendency to hypoglycemia is apparent, nourishment is left on the bedside table. No adrenal cortical extract is given up to this time.

The blood pressure is taken under basal conditions each morning, and the patient is weighed daily before eating. The patient's symptoms are watched closely. An estimate is made of the dosage of desoxycorticosterone necessary for normal hydration; edema indicates overdosage. The blood count and hematocrit level are examined on alternate mornings. Sugar and chloride levels are recorded frequently. The serum sodium and potassium are estimated if possible every two or three days. In some instances relatively stable serum calcium levels indicate the amount of hydration of the blood. Serum calcium levels should not be expected to be within normal range, however, unless the blood protein level is normal. The plasma albumin is typically low. The minimal dose of desoxycorticosterone which will produce an optimum effect is preferred.

After one or two weeks the patient leaves the hospital. Daily injections of desoxycorticosterone are continued for four to six weeks. Reexamination is made as often as seems advisable. At the end of this time pellets of desoxycorticosterone are implanted. The pellets are calculated to supply daily one-half that amount given by injection. The approximate absorption from the pellets we have been using is 1 mg. per day from each 300 mg. injected. We expect such pellets to last for eight months to a year, or more. After pellet implantation salt is given only to maintain weight, blood pressure, hematocrit level, and general sense of well-being. The Addison's patient is advised not to exert himself strenuously.

When muscle aching or other symptoms indicate that desoxycorticosterone and salt alone are not sufficient therapy, extract is added. Relatively small doses of extract under these circumstances may produce considerable symptomatic improvement. In these cases 2.5 to 7.5 cc. per day of adrenal extract (Upjohn, Wilson, or Parke-Davis) is given.

EMERGENCY KIT AND PREVENTION OF CRISIS

The patient is given written instruction on how to handle infection or crisis. Similar suggestions are given to the physician in charge. An emergency kit is particularly necessary for patients who live some distance from a hospital. The kit contains 1000 or 2000 cc. of normal saline or, preferably, Ringer's solution, and 50 cc. of cortical extract, 10 cc. of desoxycorticosterone in oil, and sulfathiazole tablets. The patient is instructed to go to bed and call the physician in charge at the first sign of infection. If the infection is any more than of the mildest sort, the patient should be placed in a hospital and treated as a case of impending crisis. Should diarrhea or vomiting, serious weakness, or drowsiness occur at any time the patient is advised to go to the hospital.

If no complications arise the patient is seen at approximately monthly intervals when a minimum of reexamination includes interval history, weight, blood pressure, and a careful blood count including hematocrit readings. We prefer, in addition, to have a record of blood sugar, chloride, sodium, and potassium.

When symptoms begin to recur, when the blood pressure begins to fall, or when weight is lost, the old pellets are removed and other pellets implanted. Desoxycorticosterone requirements are not reestimated, but an attempt is made to reduplicate desoxycorticosterone absorption, judging it from a time during the life of the pellet when the patient was reasonably well. In nearly every case so far treated we have been able to decrease the amount of desoxycorticosterone used. At present we are using doses much smaller than those of a year or more ago.

THE TREATMENT OF CRISIS

The handling of the patient in crisis is approximately as follows: With the least possible delay the patient is put to bed in a private room. He is kept warm and is examined only sufficiently to determine the presence of any serious infection; in extremis even this is omitted until treatment is begun. *If he has not been under treatment previously*, one blood sample is taken for tests outlined previously. An intravenous solution of 1 per cent saline and 10 per cent glucose is started immediately. The first 1000 cc. is given rapidly and is repeated after four hours. Ten cc. of adrenal cortical extract is given intramuscularly and, if the circulation is poor, 10 cc. is given intravenously as well. A common dose for an acutely ill patient is 50 cc. per day. The dose of cortical extract depends upon the severity of the crisis and may be as high as 150 cc. in twenty-four hours. One nurse is in constant attendance and is warned that the patient is to have nourishment every two hours or more intravenous glucose four hours after the cessation of the initial intravenous solution in order to avoid severe hypoglycemia. *If the patient has had no previous therapy*, 5 to 15 mg. of desoxycorticosterone daily may be given for the first two to three days, then 5 mg. or less per day. *When pellets are present,* the dose should be less. Rather severe edema may follow the intravenous administration of saline solution and large doses of extract, without additional desoxycorticosterone having been given, if desoxycorticosterone pellets have been implanted previously. *When desoxycorticosterone pellets are intact,* an effort should be made to prevent fall of blood potassium to abnormal levels. We prefer, therefore, if pellets are in place, to use Ringer's solution instead of saline solution and to see that as soon as possible the patient gets from 2 to 4 Gm. of potassium per day. Ringer's solution supplies 0.42 Gm. of potassium chloride per liter. Extra potassium may be given as potassium citrate in solution or as capsules in doses of 3 or 4 Gm. per day. In treatment of crisis one relies chiefly on (1) adequate quantities of Ringer's solution and glucose, (2) the avoidance of serious hypoglycemia, and (3) particularly large quantities of adrenal extract. The already reduced mortality from Addison's disease will be further reduced when other potent cortical hormones such as corticosterone can be given in adequate amounts.

CARE OF THE AGED

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In a discussion of the care of the aged, it is important to have some idea of what constitutes old age. Someone has defined it as "just twenty years older than you are." While this definition appeals to most men of my age, it is hardly exact enough to be acceptable to the collective scientific mind of this audience. Victor Hugo gave us a more clear-cut concept when he said that "forty is the old age of youth; fifty is the youth of old age." Stieglitz, in the introductory chapter of his excellent work on *Geriatric Medicine,* accepts this view when he says: "It is pragmatic to consider that the majority of the problems peculiar to geriatrics start at about forty years of age, the approximate meridian of life." He modifies this statement by calling attention to the obvious fact that individuals differ widely in the rate at which they age. Stieglitz subdivides the field of geriatrics into the periods of "later maturity" (45 years ± 5 years), senescence, and senility (70 years ± 10 years).

The increasing interest in the medical problems of old age is manifested by the number of books and articles, both popular and scientific, published on the subject within the past few years. This interest is based upon the steadily increasing proportion of older members in our population, caused by the decline in our birth and death rates prior to the present World War.

Notwithstanding the growing demand for practitioners especially interested in treating older patients, it is doubtful that many will ever limit their work to geriatrics, for at least two reasons. One is that their prospective patients would probably dislike the idea of branding themselves as senile by openly consulting a specialist in the diseases of old age. Another and stronger reason is that in order to insure the best results in aged patients, medical supervision should begin while they are in the prime of their maturity. For these reasons it is probable and desirable that, instead of a comparatively few practitioners limiting their work exclusively to geriatrics, family doctors and internists generally will become interested in conditions peculiar to old age. So far as possible, preventive medicine should be practiced in mature as well as in young and growing individuals.

General Principles

Of prime importance in caring for older patients is the avoidance of too radical changes in habits of eating, sleeping, and general hygiene. Old age is marked by involutional processes, and the aging organism adapts itself less and less readily to great changes in environment or in habits of living. In the treatment of older patients it is often necessary, of course, to alter their usual routine; but such alterations should be as gradual and as tactfully made as is consistent with one's professional judgment—and possibly with one's self respect. This dislike of change is largely responsible for the dread of hospitals that so many of the aged have; although to many a stay in such an institution is a great and happy adventure. If approached tactfully and assured that their routine will be upset as little as possible, most old patients can be persuaded to go to a hospital. Then if the nurses and house staff are kind and considerate, they may have to be persuaded to leave when the time comes.

In this connection, it may be noted that the elderly stand surgical operations comparatively well. It is really not humane to allow an old man to wear a truss or an old woman to suffer the consequences of a prolapsed uterus, when either condition could be easily and safely relieved. One of the most cruel mistakes that can be made is to let the senescent patient feel that nothing can be done because he is old.

In the two extremes of life the digestive tract is of major importance. Most of the older generation were brought up to believe in the virtue of "moving the bowels" early and often. It is seldom necessary to prescribe laxatives for these patients; they usually do that for themselves. Too many of them learn to regard a large bowel movement as an absolutely essential prelude to a successful day. Some observant wit has described the three ages of man as: (1) when he boasts of the pretty girl he dated the night before; (2) when he boasts of the good dinner he ate last night; and (3) when he tells everyone of the good bowel movement he had this morning. Most elderly people would not consider relinquishing their laxatives; but at least we can remind them of Hippocrates' advice to "Use purgative medicines . . . not without proper circumspection." (Aphorism 24).

It is just as important for the old patient to have a balanced diet as it is for the child. Perhaps the chief dietary mistake of elderly individuals, and one for which doctors have doubtless been largely responsible in the past, is to limit their intake of proteins too sharply, in the belief that such foods would cause or aggravate rheumatism, arthritis, high blood pressure, kidney trouble, or other degenerative diseases. Many senescent individuals suffer from mild or even severe protein deficiency. At least one gram of protein per kilogram of body weight should be provided daily.

The diet of the older patient, both in disease and in normal health, should be supervised enough to insure an adequate intake of vitamins and minerals. If, because of defective teeth or poor appetite, or even pure whim, the diet is not properly balanced, the deficiency should be supplied by vitamin concentrates. Most older patients like to take medicine anyhow, and concentrated vitamins are better than placebos. There is real danger of an old patient's becoming a victim of avitaminosis, particularly if he has always been a dietary faddist.

One important point is to caution the individual at middle age or beyond against excess fat. The consequences of obesity are too well known to need emphasis. On the other hand, weight should not be lost too quickly.

So far as I know, there is no valid reason why older patients, even those with hypertension, should be deprived of coffee or tea, unless they interfere with sleep. Indeed, the vasodilator effect of the caffeine may be beneficial. Tobacco may also be permitted, except for the rare individual who is afflicted with one of the vasospastic disorders; and he may be consoled by the substitution of alcohol as the vasodilator *par excellence*. Alcohol, *properly used*, is one of the greatest blessings of old age. Its food value may or may not be an advantage; but it usually has a sedative effect, and is perhaps the most satisfactory vasodilator we have. It was Lewellys Barker, I believe, who said that a man was a fool if he drank whiskey before he was 50, but a bigger fool if he did not drink after that age.

It has been demonstrated clinically and in the laboratory that the curve of hydrochloric acid usually decreases with age, and that after 60 there is often complete achlorhydria. Dilute hydrochloric acid in doses of one-half to 1 teaspoonful before or with meals is a most useful prescription for the elderly patient who is troubled with diarrhea or with gas after meals and whose tongue is dry and coated. A nursing trick worth knowing is that this acid can be given in tomato juice as a cocktail, with decided improvement in the taste of both acid and juice. It also blends well with buttermilk and with grapefruit or orange juice.

The question of exercise for the old could be the subject of a round table discussion in itself. Certainly, however, there can be little argument for strenuous exercise after 50. Even golf, generally considered the ideal recreation for the mature and old, can be overdone if taken too seriously. Light setting-up exercises which employ chiefly the trunk muscles may be beneficial. It may be apropos to recall that during the first World War the famous athlete, Walter Camp, boasted that he kept President Wilson's cabinet in prime condition by drilling them in his so-called "Daily Dozen" exercises. One member of the cabinet steadfastly refused to take these exercises. He was Josephus Daniels, who recently celebrated his eighty-first birthday by writing his usual vigorous editorials for his newspaper, the *News and Observer*. All the other members of the cabinet are dead, and Walter Camp himself died at the age of 66.

Although too much exercise may be dangerous, a certain amount is necessary at all ages, and it is generally recognized that an old patient should not be kept in bed any longer than is absolutely necessary after an illness or an operation. There is danger of hypostatic congestion which may easily lead to fatal pneumonia. The hazard of vascular thrombosis is much greater than in younger individuals. Even if he escapes these evils, an old patient who is kept in bed too long is apt gradually to lose interest in life; his appetite may fail in spite of tonics; and he may slip away so imperceptibly that it is hard to know when the loosing of the silver cord began. Many an old patient has died days after the crisis of pneumonia—when a younger patient would have been rapidly convalescing. Recently Paul White has stated that he does not keep his older patients in bed more than three or four weeks after coronary occlusion, although formerly he advocated six to eight weeks of bed rest.

The Smith-Petersen nail has been a life saver for the elderly patient by shortening the period of inactivity after a broken hip.

It is generally recognized that the old sleep lightly and require less sleep than in earlier life: "And he shall rise up at the voice of the bird." Sedatives are often necessary in older patients, but should be used cautiously—as, indeed, they should be at any age. Often the effect of hypnotics in older patients is the opposite of that desired—particularly when cerebral arteriosclerosis is present. A wakeful night followed by a morning of mental confusion may result from their use. It is often necessary to try out a number of sedative drugs before a suitable one is found. It has long been accepted that the elderly tolerate opiates badly, although in the dyspnea of cardiac failure small doses of morphine may be life-saving. An old fashioned remedy often forgotten is hyoscine. This either works like a charm or makes the patient temporarily wild; fortunately it seems to work best in the patients who tolerate opium badly.

The Endocrines

The endocrines have been a great boon to the aging, and will probably fill an increasingly large and useful place. As our knowledge of them becomes more sharply defined, their use will become more scientific and less empirical, and their abuse will be abated. The estrogens must be given first place among endocrine preparations. If we accept Victor Hugo's criterion, the menopause is the very beginning of old age for most women. Long after the actual cessation of menstruation women may suffer from what Werner has called the delayed menopause, and an estrogen may give such patients a new lease on life even ten years or more after their regular periods have stopped. While men may occasionally suffer from involutional melancholia and may be helped by injections of the male hormone, such cases are rare as compared to the number of women who need treatment.

Another condition in which the estrogens may be of great service is osteoporosis in old women.

Senile pruritis is often helped by administration of the appropriate gonadal hormone. In coronary disease the use of testosterone has been reported to be well worth while. In cancer of the prostate both castration and estrogen therapy are giving good results. A certain amount of involution takes place in the thyroid gland in old age, and in some cases distinct hypothyroidism may develop. A stubborn anemia in an old person may respond to small amounts of thyroid.

Infectious Diseases

One compensation for growing older is the acquisition of a relative immunity to many of the infectious diseases. Perhaps fortunately this does not apply to pneumonia, poetically described by Osler as "the friend of the aged." During the 1918 epidemic and since, influenza has shown a rough kind of chivalry in dealing more gently with the very old and the very young than with its more robust victims. As a general rule, infections in elderly patients tend to produce less violent reactions than in young people. The fever is lower or absent, the leukocyte count is usually lower, the prostration is more marked.

It is not often that an individual past middle age is seized by one of the so-called childhood diseases; but when this does happen, the illness is apt to be quite severe. Measles has a high mortality in old patients. Second attacks of whooping cough occur fairly frequently in mothers, grandmothers, and nurses who are exposed to it through contact with children, and may be serious in the presence of a failing circulation or of cerebral arteriosclerosis.

Prior to the advent of sulfanilamide, erysipelas was often a grave illness in aged patients; but now it has been robbed of its sting. Herpes zoster is far more apt to leave severe neuritis in the old than in the young. X-ray therapy over the affected dorsal nerve roots frequently gives great relief. It is well to remember that the virus of herpes is closely related to that of chickenpox, if not identical with it, and that a number of children have developed chickenpox after contact with an older victim of herpes zoster.

Infections in the aged are to be treated along the same general principles as in the young, but with particular caution not to employ too drastic methods. Another important point is that the older patient needs more time to recover from the effects of infectious diseases. The late Dr. J. M. Templeton, a country doctor with whom I was associated for a few months prior to beginning my internship, had one of the keenest medical minds I ever knew. Shortly before his death, when he was well beyond the three score and ten mark, he told me that if he had his professional life to live over again, the greatest change he would make in treatment would be to keep his patients in bed longer after infectious diseases. Stieglitz estimates that "for each five years of life it takes about a day longer for the body to repair and rehabilitate itself after an acute infection." I am not sure that I am ready to accept this statement, however—perhaps because I would be penalized too severely after my personal periodic attacks of influenza. It seems that an extra day of bed rest for each ten years should be enough, since the danger of thrombophlebitis and of hypostatic pneumonia must be kept in mind. While in bed, the patient should be turned often, and encouraged to move the limbs from time to time, and to breathe deeply at frequent intervals.

The sulfonamides have revolutionized the treatment of many of the infections; but, like most powerful weapons, they should be used cautiously. Because the bone marrow is more easily depressed in the elderly patient than in the young, the blood counts should be followed carefully. I have an impression, which I could not prove, that since the sulfonamide era more older patients with abnormally low white cell counts have been seen. Most of us have acquired a wholesome respect for the power of the sulfonamide drugs, and have learned to restrict their use to sharply defined conditions. In the early days of sulfonamide therapy the wisecrack was current that in a certain medical center every patient admitted with fever was given sulfanilamide for three days, and that if he still had fever then, he would be examined.

THE DEGENERATIVE DISEASES

With the flight of the years, as the danger of infections becomes less, the degenerative diseases—the result of wear and tear—become more prominent. The most important effect of aging is upon the blood vessels. They gradually lose their elasticity and their lumina become narrowed. This vascular sclerosis in turn interferes with the nutrition of the various parts of the body: the brain, the kidneys, the bones, the muscles, the gonads, the pancreas, the skin, and even the heart itself. A gradual increase in blood pressure is usual. The more marked the degenerative process in the kidneys, the more rapidly progressive is the hypertension, and the more productive of symptoms.

The real cause of high blood pressure is still unknown, in spite of a vast amount of speculation and research. Its prevention is still on the knees of the gods. At present we have done all we know to do when we see that our elderly patient gets sufficient rest and relaxation; that his diet contains enough vitamins and minerals and not too many calories; that his weight is optimum; that no obvious foci of infection are retained; that there is no urinary obstruction; that he has enough interests to insure him against boredom; and, finally, that he is taught, so far as possible, to adopt Osler's Way of Life—to live only one day at a time.

The effect of the arteriosclerotic process upon the coronary arteries is too well known to require elaboration. Fortunately, with increased recognition of coronary disease, it is being robbed of much of its dread. No longer is it necessary for the patient to feel that he has heard his death sentence; enough of his neighbors and business associates have lived with heart disease for many years to give him hope. It is important to explain to its victim the mechanism of angina, simply and clearly enough for him to realize that when he feels pain on exertion he should stop immediately until the pain has subsided. The action of nitroglycerin can also be described, and the patient should be assured that it is as harmless as it is effective; that it never creates a habit or "wears out"; and that it should always be used in case of doubt. The patient should, of course, be instructed to stay within the limits of his exercise tolerance.

The effects of cerebral arteriosclerosis are also well known. Early symptoms include dizziness, fatigue, a tendency to be drowsy, failing memory (especially for recent events), and irritability and depression. In advanced cases the mental symptoms become more severe, until the so-called senile dementia is reached, with marked changes in character and conduct. At this stage, medicolegal complications may arise from the impaired judgment of the patient, and it may become necessary to declare him incompetent.

The intriguing observation has been made that in some cases the degree of organic change in the brain by no means parallels the personality changes in the patient. "Recent studies have indicated that in the past altogether too much stress has been laid upon the organic factors in the mental disorders of advanced years and not enough on what may be termed the psychological hazards of aging. Some patients who have shown rather profound mental changes attributable to advancing years have been found at autopsy to show astonishingly little pathology of the brain; whereas, on the other hand, patients in whom rather striking cerebral lesions can be demonstrated have shown an extraordinarily good ability to adjust and to meet the demands of their environment."

Alvarez has repeatedly called attention to a phenomenon that occurs fairly frequently in the elderly patient, in which the symptoms are referred to the digestive tract, although the cause is in the brain. He says that when a middle aged or elderly patient suddenly has an attack of "indigestion," with pain or discomfort in the upper abdomen, nausea with or without vomiting, and subsequent loss of appetite, the real cause is most apt to be thrombosis or hemorrhage in a cerebral artery. On careful inquiry, the history of dizziness and possibly headache at the time of the attack, and often a personality change afterward, will usually be obtained. The cerebral accident frequently does not cause paralysis, and may easily be overlooked.

The treatment of cerebral arteriosclerosis, like that of arteriosclerosis in general, is not very satisfactory. However, it is possible to begin in mature or even early adult life to prepare for it, and possibly to prevent some of its manifestations by cultivating a variety of interests, and by building up a well integrated personality. As Overholser has well said, "Far more important than the organic changes is the functioning of the various organs, and even more so the functioning of the personality as a whole. The well-integrated personality . . . may survive . . . the ravages of cerebral arteriosclerosis."

The waning of the sexual power in the old is really a conservation process on the part of nature. Except for those devotees of the French school of novelists who make sex their god, the aging man may be induced to look upon this as philosophically as did Sophocles in Plato's *Republic,* who, when asked "How does love suit with age?" replied, "Peace; most gladly have I escaped the thing of which you speak; I feel as if I had escaped from a mad and furious master."

In the old man the prostate is far more apt to cause nocturia and polyuria than are the kidneys. Fortunately prostatism is now much more easily and safely relieved than formerly. The high incidence of cancer of the prostate, and Osler's admonition never to overlook a rectal examination, should be kept in mind.

A mild form of diabetes often results from arteriosclerotic changes in the pancreas. Often it yields to a restriction of the carbohydrates, without the use of insulin; and in the obese individual this dieting may be beneficial. However, such a patient should be followed closely enough to detect an increase in the diabetic condition. The old patient may be given the assurance that diabetes is much less severe at his age than in a young person.

The word "arthritis" is apt to bring terror to almost anyone. According to X-ray evidence, a certain amount of arthritis is universal in old people. This does not mean, however, that all old people will be crippled or seriously disabled by it. Virtually all authorities on the subject agree that the severe, crippling form of atrophic arthritis begins in the first half of life and that new cases are rare after 60. The hypertrophic or senile type is present in every individual after 60, but this form rarely completely disables.

In the treatment of either form of arthritis a proper balance between rest and exercise is essential. Goldthwait, who was one of the pioneers in the treatment of arthritis, gives fatigue as the most important single cause. The corollary of this statement is the therapeutic value of rest. After the acute stage has subsided, graduated exercise should be used. Heat to the affected joints is also beneficial. If the patient is obese, gradual reduction should be advised. As for drugs, aspirin is perhaps the best and safest pain reliever. Another old stand-by which is too often forgotten is iodine. Of supreme importance is reassurance. If convinced that he will not be hopelessly crippled, many an old timer can forget the pain and stiffness of his joints.

In the aged, as in the growing child, there is often a tendency to anemia. In the child, this is due to the excessive demands upon the bone marrow occasioned by growth; in the old person, anemia results when the marrow is not equal to the relatively moderate task of supplying blood for the daily needs of the body. Routine blood counts should be made at intervals, and anti-anemic therapy instituted when indicated.

Psychological Factors in Old Age

In dealing with the aged, it is of the utmost importance to maintain their morale, so far as possible. We need to be sympathetic but not sentimental. Stuart Chase, in *The Road We Are Traveling,* expressed a profound truth when he said: "Man is a working animal, as any biologist will tell you. They want to *belong*: to feel that they are a part of a living community, that they have a place in it which other people recognize." Many of the most unpleasant traits of adolescence are due to the desire, conscious or subconscious, on the part of the youngster to make a place for himself in the community, and to be recognized as belonging. Just so, many of the most unpleasant traits of old age are due to the desire to continue to belong, and to hold to a place in the community. Every normal human being, young or old, wants to feel that he has a definite place in the world and that he is important. As long as an old man is capable of continuing his occupation he is happier if allowed to do so. Even after the age of retirement is reached, and he is compelled to relinquish his job to some younger individual, a definite task around the house should be assigned him, and he should be made to feel that he is needed for that particular job. The natural tendency of the elderly to become egocentric is greatly increased in the person who has not prepared for old age by cultivating a number of outside interests, and who has nothing to do but to sit and think about himself.

One of the possible blessings of the present war is a halt and perhaps a reversal of the trend in industry to exalt youth at the expense of age. Employers are learning that most men are capable of working many more years than was formerly thought profitable, and that the steadiness and judgment that come with experience largely make up for the greater strength, speed and enthusiasm of youth.

If it becomes necessary, for economic or other reasons, for an old couple or widowed mate to live with a younger family, it may be necessary to let it be known at the beginning that such an arrangement must not disrupt the happiness of the family. Even if feelings must be wounded, the member of the older generation should not be allowed to come between husband and wife or between parents and children. The old man or woman too senile to see the reasonableness of this position would probably be just as happy in a home for the aged.

Stieglitz comments that wisdom depends upon experience, in which time is a major factor. Aging does not create wisdom, but "merely permits it to develop in those intelligent enough to learn by experience and prepared to observe and profit by such learning. . . . The central nervous system of a body politic is composed of older men and women, just as the cells of the brain are among the oldest in the body of the individual organism."

This observation recalls Walter Lippman's recent characterization of Winston Churchill: "He is the one certainly authentic example of greatness in a public man who moves among us . . . when men age they may grow vain, irascible and self-centered. Or they may grow wise, benign, compassionate, and universal. Churchill has aged well."

This tribute reminded me of a medical statesman, a recent president of the American Medical Association, who has also aged well. At 75, Dr. Nathan B. van Etten, as he steered the greatest medical group in the world through one of its most critical years, inspired all who came in contact with him by his magnanimity, his wisdom and his greatness.

It is not given to many to be van Ettens or Churchills, but at least such examples should inspire young, mature and aging persons alike to strive to grow old gracefully. If "the child is father of the man," then the mature man must be the prototype of the old man. Plato said many centuries ago: "Old age has a great sense of calm and freedom; when the passions relax their hold, then, as Sophocles says, we are freed from the grasp not of one mad master only, but of many . . . He who is of a calm and happy nature will hardly feel the pressure of age, but to him who is of an opposite disposition youth and age are equally a burden."

SUMMARY

1. The habits of the old should not be changed too rapidly or too drastically.
2. An adequate diet should be maintained, but excessive weight avoided.
3. Neither exercise nor rest should be overdone.
4. The endocrines, especially the estrogens, are often helpful.
5. The frequency of infectious diseases decreases with age.
6. The degenerative diseases assume increasing importance with advancing age.
7. The best preparation for old age is the cultivation of a variety of interests.

WAR SURGERY — 1943

THE SPEAKER: *Gentlemen, we have in our midst a very distinguished guest, Major General W. H. Ogilvie, of London, England, Chief Consulting Surgeon, British Army of the East, who will talk to us on some of his war experiences.*

MAJOR GENERAL W. H. OGILVIE: Gentlemen, I feel greatly honored by being allowed to speak to you today. My choice of subject is unfortunately limited by my experience. For three of the last four years I have been cut off from books, from the stimulating influence of cold civil surgery, from the great centers of research, and most of what I know of more recent advances has reached me in communications from friends.

I have, however, been privileged to see the treatment of the wounded in three of the main theaters of war differing widely from each other in all essentials, and to hold the post of Consultant Surgeon during two spectacular campaigns, the advance from Kenya to Addis Ababa and the two-thousand-mile chase from Alamein to Tunis. In each I had amongst my junior colleagues some of the most gifted young men in British surgery and pathology. We have never been able to study our material with the leisure that the pure scientist demands, but in so far as research means constant inquiry we have done our best to investigate the puzzles that came before us and to submit each new suggestion to the test of critical and impartial trial.

I have always held that the primary treatment of wounds is the most important part of war surgery. If it is good, all that follows is simplified; if it is bad, no amount of subsequent skill will make up for it. If, then, forward surgery is the key to everything that follows, the place of the Consulting Surgeon during a battle is with the Army. That, at any rate, has been my theory and my practice, so I shall not expose myself to criticism by attempting to give you a scientific address, but will confine myself to a few remarks based on my personal experience.

The first thing I want to say is that the teaching of experience is applicable in its entirety only to the particular time, place and circumstance in which that experience was gained. Each battle brings a new terrain, different weapons, fresh supply problems, methods on trial for the first time; each battle demands, therefore, not fresh ideas, but a reconsideration of the old and the new together, and an ability to judge fresh needs wisely. Knowledge is filed experience; wisdom is experience filed on the loose-leaf system.

The battles of the 8th Army have given us opportunities to perfect surgical organization and to study modern methods in the treatment of wounded. The lulls between the battles have enabled us to call surgical conferences and to exchange information with each other.

There are four things that any surgeon, however brilliant and however experienced he may be, must learn afresh when he comes to a theater of war:

(1) That military surgery is not just traumatic surgery in uniform; it is part of the science of warfare.

(2) That the clinical picture of a wound has to be learned afresh, just like the signs and the symptoms of appendicitis.

(3) That no surgical procedure in war is complete in itself but is part of a process whose preceding and subsequent steps must also be taken into account.

(4) That the teachings of the last war, which still dominate articles and textbooks, are not necessarily applicable today.

The aim of the command in any civilized army is to give the casualties the best possible treatment. When anything is allowed to interfere with military efficiency, the lives of the well and the wounded are risked together. Efficiency in modern war means speed, simplicity and concealment.

The more elaborate the surgical outfit, the longer does it take to set up, and (more important in mechanized warfare when the first warning to go often comes from the guns of an approaching tank) the longer does it take to pack. An elaborate unit takes more transport, and in the battles of today, especially at times of rapid movement, like our two-thousand-mile chase after Rommel, every lorry that can be kept off the roads and every gallon of petrol that can be saved for fighting vehicles is of life and death importance. A large surgical group is a conspicuous target from the air, and to hostile reconnaissance aircraft it means expected casualties and therefore preparations for an attack. Even so simple a precaution as a call for volunteer donors among reserve units spells zero hour within a week to those many enemy agents who abound among the civil population.

Perhaps the most important thing, therefore, that we have learned is to make our methods and our units as simple as we can.

The clinical assessment and examination of wounds caused by modern projectiles is an art that can only be learned by the side of the stretcher. Experience alone can tell which are innocuous, which are dangerous, and in what direction danger lies.

Many of the worst mistakes made by the expert surgeon new to war work are due to his failure to realize that he is not the arbiter of a patient's fate but simply on the production band where many work at the same chassis. He must do not what he thinks to be best but what he knows to be safest. Many methods that are ideal when the patient can be kept at rest and in good surroundings and under observation of his own surgeon may go seriously wrong if he must be evacuated at once with a rough journey and an uncertain destination in front of him.

The surgery of the last war culminated in the practice of prophylactic excision, an operation based on the assumption that in wounds reaching the surgeon within six hours infection could be prevented by the painstaking excision of all foreign material and damaged tissues. This practice is still preached widely today. Why do we do it, and is it necessary? Débridement was good under conditions of the last war, that is where a steady flow of wounded reach a surgical center within six hours in numbers that do not impose additional delay, where there are well equipped theaters, good lighting, clean, dust-free air, and an abundant supply of water and sterile materials. Then débridement is good. But these conditions are seldom found in modern war. It is not necessary, for wide excision of all suspect tissues is a time consuming and damaging procedure and only justified if it is to be followed by primary suture. It is our experience that the dangers of 1918 can be averted by simpler means.

All these are simple lessons, but they are hard to learn. All fresh surgeons coming to a battle for the first time make the same mistakes. The higher their standing in civil life the more difficult they are to teach. The following are the seven cardinal sins of war surgery, and among them I do not include primary suture and immediate evacuation of fresh wounds because that is worse than a sin; it is a crime for which the only remedy is dismissal.

(1) Unnecessary operations on through-and-through bullet wounds of soft parts. The majority of these with rest and sulfonamide drugs heal rapidly and leave no disability. Operation means loss of time and loss of function.

(2) Removal of skin. Skin is, of all structures, the most viable, the most resistant to infection, and the most irreplaceable. It may be split to give access to deeper structures, but it must never be excised except as in the edges of the head or abdominal wounds as a preliminary to suture. Cutting around of entry and exit wounds is the mark of the beginner.

(3) The wide excision of muscles, especially of the transverse section of normal muscle groups, in order to expose a deeper tract. The subsequent disability is shocking and quite unnecessary. Trimming, drainage, and sulfonamide drugs give greater safety.

(4) Insufficient slitting of fascia. While excision is destructive, relief of tension is life saving. The tough fibrous sheaths of thigh and calf should be divided freely when a deep lacerated wound is being trimmed.

(5) Tight plugging. I have never met the surgeon who admitted to this and I have never met the surgeon who did not do it at some time or another. A man whose wound has been plugged arrives very ill and only recovers when the deep pus is uncorked by pulling out the gauze that retains it. The only safeguards of plugging are free incision in muscle planes and loose laying in of a single sheet of vaseline gauze.

(6) Unsplit plasters. Closed plasters give trouble so seldom in hospitals, as we all know, that surgeons find it very difficult to realize their dangers in transport. An unpadded case owes much of its value to the control of circulation given by even pressure. But jolting in an ambulance or an airplane may start small hemorrhages or swelling in a recently injured limb, harmless in itself but sufficient in the closed case to lead to a dangerous increase in tension. We have been forced to issue an order that every lower limb plaster must be split before evacuation, and we are tending to abandon the unpadded case altogether and to use padded plasters or standard methods of fixation like the Tobruk plaster for the lower limb and the Alamein or thoracobrachial plaster for the upper limb.

(7) Unnecessary mutilating operations for gas cellulitis. Infection of subcutaneous or of connective tissue planes by gas forming bacilli, Colstridia, has been fairly common, particularly since the campaign in Sicily and Italy; but gas gangrene has been relatively rare, and not often leading to loss of life or even necessarily of a limb. The reasons for this are many and to discuss them now would take too much time, but two things are important: First, every for-

ward surgeon should know the kind of injury that is liable to be followed by gas gangrene, and that is the injury of the large muscle groups complicated by injury to a main vessel. He should give the patient a prophylactic dose of serum and flag his documents so conspicuously that the danger is appreciated at each surgical center that he reaches. Second, all surgeons should know that gas itself is the least important sign in gas gangrene. Pain, a rising pulse, a "deadhouse" smell, and solid edema in the limb, mean very much more. Today when so many cases of gangrene recover even after drainage or excision of muscle groups, no surgeon should amputate a limb for this purpose, if he can possibly discuss the prognosis with a more experienced man.

I have already taken much of my allotted time over these simple matters. Forward surgery must be simple. We developed a group of very small and very mobile surgical units, each carrying only one surgeon and an anesthetist and the minimum of equipment. These units formed a fluid surgical reserve at the disposal of the Commander of the Army —to be used by him in any combination to reinforce the forward units according to the tactical needs of the moment. The surgeons in these units were the pick of our forces, the best men we could find, and they were changed at frequent intervals. During their periods out of the line they were given refresher courses in special branches of surgery.

The only type of specialist unit which we found necessary to put in the actual fighting zone was the neurosurgical unit. We had an advanced section working in combination with a blood team and an ophthalmic unit at an advanced clearing station, but far enough back to tap all the main lines of evacuation; and also an advanced base hospital section taking the operated cases sent through from the advanced section and the lighter cases that were not done forward.

The surgery of abdominal wounds with the type of surgeon that we picked for forward work has not been regarded as a specialty. Courage and decision at the time and retention afterwards are the key to recovery.

Maxillo-facial surgery is not front-line work. The less that is done beyond fixation of jaw fragments until the reaction of repair is over, the better will be the subsequent repair.

Major thoracic surgery again should not be undertaken in the battle zone, for large thoracotomies are rarely needed and they are very fatal when done under unsatisfactory conditions. Most cases travel well after rest, morphia, oxygen, and the mechanical correction of sucking wounds or tension pneumothorax.

Fractures require wound toilet forward and should be transported, at the earliest opportunity, to a fracture center.

Well, gentlemen, in September we captured a German colonel who talked more than most, and when we asked him what he thought of the war he said, "If we had British food, American equipment and Italian enemies we should like it." I should like to finish by a small tribute from the 8th Army in the Middle East to American equipment—unfortunately the only form of American cooperation that we enjoyed because your soldiers were not on the eastern side of Tunis. We felt that the battle of Alamein we owed partly to General Montgomery and very largely but partly to the arrival of Sherman tanks, and these special cases I have discussed which had to be sent back owe very much not only to the skill of your airmen but to the designers of machines like the Douglas and to the overwhelming production of your factories.

While we now clearly have cooperation between us, I think we can say that victory is in sight and I am sure that after victory the perpetuation of a peace which is secure and lasting depends above all on friendship and trust between our two great countries.

Surgery of the Pancreas

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There have been definite and real advances in the surgical treatment of diseases of the pancreas within the last few years. Some of you may have some question in your mind as to what actually constitutes surgery of the pancreas. In the past, it was confined largely to the treatment of acute pancreatitis with perhaps an occasional operation for a fistula or for a pancreatic cyst.

Shall we briefly outline the history of the radical treatment of tumors of the pancreas? As early as 1907, two French surgeons described operations, based on their anatomic studies, which permitted removal of portions of the pancreas and of the duodenum. It remained, however, for a German surgeon, Kausch, to perform the first radical pancreatoduodenectomy for carcinoma in this location, in 1912.

Following the discovery of insulin, Harris described the clinical syndrome of hyperinsulinism, even before the discovery of a benign, hyperfunctioning tumor of the pancreas, and stated that such a lesion must exist. It remained for Wilder to discover such a clinical case, and for Dr. W. J. Mayo to remove this tumor in 1927. Two years later, Dr. Roscoe Graham removed the first benign tumor, since the one removed by Dr. Mayo was a definite carcinoma.

Previous to 1935, there were a few sporadic attempts at radical removal of tumors in this location. Interest was awakened by the report of Whipple, Parsons and Mullins of the successful two-stage removal of a carcinoma of the ampulla of Vater. The same year, Hunt and Budd were able to collect 76 cases in which a malignant lesion had been removed from this area, but most of these had been mere local excisions, usually by a transduodenal operation.

To realize how much interest has been evoked concerning the treatment of tumors of the pancreas, one needs but to call attention to the book, "The Surgery of Pancreatic Tumors," by Brunschwig of this city, which was published last year.

Since the surgical diseases of the pancreas are not confined to tumors, let us consider the diagnosis of the different conditions that may require surgery. The symptoms should be divided into, first, those accompanying acute conditions, chiefly pancreatitis, and secondly, those accompanying the chronic conditions, cysts and other benign lesions, as well as carcinoma of the pancreas. Unfortunately, in a surgical disease of the pancreas, there are no clear-cut symptoms which direct the attention to this organ, but in acute pancreatitis there is an acute onset accompanied by shock, prostration, fever, abdominal pain, back pain, particularly in the upper lumbar region, and occasionally a mild degree of jaundice. The condition is recognized as an abdominal emergency, and it then behooves one to differentiate acute pancreatitis from perforated or penetrating peptic ulcer, obstruction of the upper small intestine, and other acute diseases, particularly cholecystitis.

In the chronic cases such as a benign, nonfunctioning tumor or cyst, there is merely the physical sign of tumor and no symptoms.

In a review of 74 of our own patients with a malignant condition of the pancreas, Kiefer found that 26 had no jaundice. In the past we have depended too much upon the early finding of jaundice to make the diagnosis. No doubt the reason these 26 patients did not have jaundice early is because the malignant condition occurred at some distance from the ampulla of Vater where obstruction of the main biliary duct would occur.

In these 26 patients the most conspicuous symptom was back pain, usually about the twelfth thoracic or first lumbar region. This pain is made worse by lying down. Perhaps the only way it can be eased is to have the patient sit up and forward to relax the anterior abdominal musculature and thus relieve tension. It is not relieved by any other measures, such as food, as is the case in peptic ulcer.

The second important symptom is that often associated with cancer of the stomach—anorexia. The loss of the desire for food, the loss of appetite, is very conspicuous in these cases.

A number of indefinite symptoms can be enumerated, some of which could be listed under dyspepsia or indigestion. Sometimes nausea and vomiting are present, and sometimes diarrhea, constipation or a foamy stool may occur if there is sufficient interference with the flow of external pancreatic secretion. General weakness and weight loss are observed.

Jaundice occurred in 48 of 74 patients and was usually painless, certainly painless during the latter stages of observation, although one might be deceived by the onset of jaundice with an attack of pain. The pain is chiefly discomfort and not the colicky sort associated with calculous obstruction of the common bile duct. The jaundice tends to be progressive and associated with many other symptoms previously described as occurring in the patients without jaundice.

What means can we use in establishing the diagnosis when the symptoms are so indefinite? In the physical examination, the demonstration of a mass is common with the benign tumors or cysts of long duration. The presence of an indefinite fullness with localized tenderness to that area is common with the acute symptoms associated with acute pancreatitis. There may be spasm, and at times if the tumor is large there may be costovertebral tenderness.

In the laboratory studies in our own cases, we have been disappointed in the determination of the enzymes of the blood. As Whipple has stated, in the acute cases it may take three or four days to get any marked concentration of blood amylase. We have not found the determination of the blood lipase or amylase of much value in establishing either the acute or chronic conditions. The estimation of the bilirubin and of the general blood picture is of importance since hemolytic jaundice must be differentiated in these jaundiced patients.

The use of the barium meal and the barium enema is of help in some cases. If there is a widened sweep of the duodenum, that is, a greater distance between the pyloric end of the stomach and the fourth portion of the duodenum, this may be due either to a benign tumor, a malignant tumor, or acute pancreatitis. In the presence of cysts and larger benign tumors or semisolid tumors, the barium enema and the barium meal may show displacement of the stomach upward, the colon downward, or any other type of displacement. For instance, with the stomach forward, we would suspect a tumor in the region of the pancreas. However, this information is inferential, and it is only by abdominal exploration that location of the tumor in the pancreas can be determined.

In chronic cases, a biopsy specimen is of dubious value, unless a positive report is obtained. However, in a study of pancreatic biopsies in cases in which chronic pancreatitis or tumor was suspected, there has been a 50 per cent error in diagnosis, so we have not depended upon biopsy in these cases.

If we have demonstrated a surgical condition of the pancreas or we think one exists, what is the surgical approach and surgical treatment? If we wish to obtain a biopsy specimen of the pancreas or to remove a small tumor from the upper portion, the approach can be made through the gastrohepatic omentum where a large portion of the pancreatic body can be visualized. However, if the location of the tumor is doubtful or if any radical procedure is required, the best approach is through the gastrocolic omentum either by severance of the anterior two layers or, preferably, by detachment of the omentum from the colon itself. By this means an exposure of the entire anterior surface of the pancreas from the head to the tail can be obtained.

The main problem relative to the surgical extirpation of various conditions of the pancreas has to do with control of the blood supply, both arterial and venous. A recent report by Pearson in "Surgery, Gynecology and Obstetrics" has given us a very clear picture of the arterial blood supply of the pancreas.

In resecting the pancreatic head, one must control the gastroduodenal artery which comes off the hepatic artery and gives off an anterior and posterior superior pancreatoduodenal artery. Likewise, there are two important arteries coming up as the first branches of the superior mesenteric artery, the inferior pancreatoduodenal branches. There are many additional arteries that come from the hepatic, celiac and the gastro-epiploic arteries, that come in from both the superior and inferior surfaces of the pancreas.

The main problem with regard to the venous circulation is the avoidance of injury to the portal vein. This vein begins directly behind the neck and head of the pancreas, from the junction of the splenic and superior mesenteric veins. It is necessary before getting too far along in any radical procedure in this location to dissect it free and to be sure that it is not involved by a malignant process. This is fortunately possible technically and in only 1 case have we found it necessary to remove a portion of the wall of the portal vein.

To go back to acute pancreatitis, unfortunately we do not know its cause, although there are a number of theories. For this reason, we are uncertain of the proper treatment. In the past, I believe, most of us have operated upon all cases of acute pancreatitis. Today, fewer and fewer patients with acute pancreatitis are being operated upon, most of them being treated expectantly.

Four causes may be considered, namely, (1) a proteolytic breakdown with autolysis of pancreatic tissue; (2) regurgitation of bile; (3) regurgitation of small intestinal contents into the pancreatic duct, and (4) infection.

Accepting one or more of these hypotheses concerning the cause of acute pancreatitis, we should direct our surgical treatment on that basis. We divide our cases into the acute interstitial, the acute hemorrhagic, and the suppurative. It is unnecessary to operate upon patients with acute interstitial pancreatitis since there is little that can be done other than to establish a diagnosis. Incision of the pancreas or the pancreatic capsule is a mistake, and drainage of the biliary tract probably does little or no good. In the acute hemorrhagic form we have felt that prolonged drainage of the common duct by T tube is probably of some benefit. This drainage should be continued for approximately three months. In the acute hemorrhagic form we do not incise the pancreatic capsule, but we do put a drain to the pancreas through an opening in the gastrohepatic omentum. With suppurative pancreatitis, however, there must be adequate surgical drainage, and it probably is well to drain the pancreatic region both through the gastrohepatic and gastrocolic ligaments.

The mortality of acute pancreatitis is from 20 to 50 per cent, depending upon the number of suppurative cases in any particular series. Our own mortality in cases reported a few years ago was 22 per cent. There is a high delayed mortality in suppurative pancreatitis, and death may occur in spite of adequate surgical drainage, as long as three to five weeks after onset. It is due to an extensive infection and cellulitis in the retroperitoneal region.

There are two additional conditions of the pancreas that at times have been considered surgical—chronic pancreatitis and pancreatic calculi. We find it difficult to be sure of the diagnosis of chronic pancreatitis. In the past the diagnosis of chronic pancreatitis has been made at the time of abdominal exploration, and many indefinite symptoms have been associated with the condition. We think there is no place for surgical treatment in this condition, and rarely for surgical treatment of pancreatic calculi.

Trauma of the pancreas in civilian life is rare. With the present war wounds and their treatment, much will be learned with regard to pancreatic trauma. Treatment may be immediate or delayed. Immediate treatment includes (1) the relief of shock, which is true of any acute abdominal trauma; (2) stoppage of hemorrhage, and (3) drainage if necessary.

Delayed treatment usually is concerned with a persisting pancreatic fistula. Under traumatic conditions, one could consider the pancreatic fistulas that occur as a result of various surgical procedures on this organ. In our experience, most of these, if treated conservatively, will close either by the spontaneous development of an internal pancreatic fistula into the intestine or by closing by fibrous union in the tract. Dr. Lahey reported the transplantation of a pancreatic fistula in 1937.

Cysts of the pancreas are uncommon, but may be encountered at any time. The diagnosis is based, first, on the presence of a tumor without other symptoms, and secondly, on displacement of organs in the region of the pancreas as visualized by roentgenologic means.

The treatment of pancreatic cysts is excision if possible. They do not tend to become malignant, but produce symptoms by pressure and extension. If the cyst cannot be excised because of size or involvement of areas in which resection is unwise, partial excision of the cyst wall can be done, leaving the posterior part in place. Careful inspection of the remaining cyst wall may disclose small pancreatic ducts, which can be cauterized or sutured with nonabsorbable sutures.

Finally, if one feels that extirpation of the cyst is unwise, marsupialization can be carried out. This probably entails the least risk. The cyst is unroofed and the wall brought up as much as possible and brought out through the anterior abdominal incision. The cavity can be loosely packed. If a pancreatic fistula persists in one of these marsupialized cysts, a secondary operation can be conducted which will transplant into the jejunum the fistula that was thus established.

The presence of a benign tumor of the pancreas, as I already have mentioned, was considered as a possibility by Harris in 1925, and such a tumor was first removed in 1929. In November, 1942, Whipple reviewed the experience of the Presbyterian Hospital, New York City, and found that in this one institution 22 hyperfunctioning adenomas had been encountered up to that time. He collected a total of approximately 100 from the literature. Since that time, including other cases not reported up to November, 1942, approximately 120 hyperfunctioning pancreatic tumors have been reported. In our Clinic we have had 3, all of which were operated upon by my associate, Dr. Marshall. These patients were all seen within a period of six months, in spite of the fact that we had been on the lookout for the condition for several years.

One of these patients, reported by Dr. Allan, was 35 years of age and had a three year history of attacks. These attacks consisted of hunger, dimness of vision, aphasia and partial loss of consciousness, so that there was frequent mental confusion. The blood sugar at one time was 66; at another time, 40; and during a severe attack was as low as 35. The one important thing in the preoperative observation of these cases is a low blood sugar, which has been observed on more than one occasion accompanying an attack.

The tumor was visualized at operation and was removed, and because of the presence of a questionable malignant lesion, the distal one-half of the pancreas was excised with the tumor intact. The slide shows evidences of invasion, a low-grade adenocarcinoma of the pancreas in a hyperfunctioning adenoma (1.5 cm. in diameter).

I would like to speak briefly now of the surgical treatment of malignant tumors of the head of the pancreas, the duodenum and the region of the ampulla of Vater. In order to effect a cure in a tumor of the ampulla of Vater (slide), we believe it is necessary to remove the prepyloric portion of the stomach, the lower portion of the common duct, and at least three portions of the duodenum, preferably removing all four portions of the duodenum, the first portion of the jejunum, and the head of the pancreas.

If there is a carcinoma of the head of the pancreas, it is necessary to remove a considerably larger portion of the pancreas in order to give the best chance of eradication of the lesion. We would remove the same amount of gastrointestinal tract and common duct.

In a general way, this slide shows what we finish up with after radical pancreatoduodenectomy: an anastomosis of the jejunum to the stomach in an antecolic position, an anastomosis of the jejunum to the pancreas, anastomosing the pancreatic duct, and anastomosis of the biliary tract to the jejunum. If the anastomosis of the jejunum to the stomach is above the point of these two anastomoses, an entero-enterostomy is done. To close the pancreas, non-absorbable sutures are used, and the blood supply is carefully controlled.

This shows the type of anastomosis of the biliary tract to the jejunum, joining the end of the common duct to the jejunum (slide). This is always done in a one-stage operation. If a two-stage operation is done, the gallbladder is anastomosed to the jejunum at the first stage.

This shows the closure of the duodenum when only the three portions of the duodenum are resected in certain early carcinomas of the ampulla of Vater; the closed fourth portion of the duodenum draws under the superior mesenteric vessels (slide).

In a patient resected by me in 1939, a mortality resulted because of a persisting pancreatic fistula (slide). For this reason, in the last few cases a direct anastomosis of the pancreatic duct or duct of Wirsung to the jejunum has been done. This is the afferent jejunum. An incision is made in the serosa, and the seromuscular coat is pushed back. A transfixion suture of silk is passed through the ligated duct of Wirsung, and the pancreas is anastomosed to the posterior wall of the jejunum. This necrosing suture of silk is then passed through the jejunum similar to the Coffey technic of ureteral transplantation, and the anterior surface of the pancreas is joined to the jejunum to complete the anastomosis.

I wish to show you a few of the specimens removed by radical pancreatoduodenectomy (slide). This is the first patient operated upon, and you can see the pyloric end of the stomach, the entire head and beginning neck of the pancreas, and the four portions of the duodenum. This shows the mucosal surface, the entrance of the common bile duct, the entrance of the duct of Wirsung, and here at the ampulla of Vater is an adenocarcinoma with a separate entry of the duct of Wirsung, the duct of Santorini and the common duct.

This is a second radical resection showing the pancreatic head removed with only three portions of the duodenum (slide).

This shows the interior of the same case with a small carcinoma of the ampulla, with every chance for cure (slide).

This man had a biliary obstruction of six months, and a two-stage operation was done (slide). This is the pyloric end of the stomach, the whole pancreatic head, the uncinate process, the pancreatic neck, and part of the body.

This is the last case that I wish to present to you —a man, 72 years of age, with a carcinoma of the ampulla (slide). I think the thing that will impress you in each of these tumors in the ampullar region is the relatively small size and, most important, the fact that there is little or no evidence of malignant spread.

To review our experience with radical pancreatoduodenectomy, in the last fourteen months we have resected 12 carcinomas in this region. There have been 2 deaths. One death occurred seven weeks after operation from pyelonephrosis, and another death occurred from surgical shock the day after operation. One patient subsequently died from recurrence of carcinoma of the pancreas. The others have continued well over this short period of time. These 12 resections were out of a group of 33 patients operated upon during the fourteen months for carcinoma in this region. One can now appreciate that a considerable number of tumors in this location are amenable to surgical extirpation, since during the fourteen month period, 37 per cent of all cases were submitted to radical resection.

Coordinating Medical Problems with Tank Warfare

By DANIEL L. BORDEN, M. D.
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Colonel, Medical Corps, Army of the United States,
Commanding Officer, Station Hospital, Fort Eustis, Virginia

Mr. Chairman, General Kirk, General Ogilvie, Ladies and Gentlemen:

Co-ordinating instruments of war with man and his environment is so essential that it is a priority in the designing and construction of tanks. The tank, with its engine of power, its blood line of oil, its temperature control by circulating water, its intake of gasoline and its exhaust of fumes, is a running machine comparable to the human body. But human hands must supply the one lacking feature that controls the nervous system of the mechanical tank. Tanks are not supplied with the special senses of man. Man, to control an active tank, must not only exert to full capacity his special and muscular senses but he must have complete control of these senses under the most favorable conditions. Tanks run and operated by man must be designed and constructed to enhance the strength of man and to minimize any of his fighting weaknesses. The Ordnance Department is well aware of this fundamental principle. To the credit of this Department there has been established a working liaison between its Development Branch and the Medical Department of the U. S. Army. Our Ordnance Department with this understanding working basis has developed the best tanks in the world. As will be shown, this fact, actually and psychologically, has a high value in the fighting qualities of our tank crews.

The Ordnance Department is constantly faced with the problem of incorporating improvements in their tanks that will co-ordinate the technical difficulties of the tank with the physical reaction of the crew. Three basic principles enter into the design and construction of a tank: firepower, mobility, and protection. Basically, these three essential details must be supported by reliability, simplicity, and accessibility. All of these basic requirements have been met in our modern tanks and we are now facing the necessity of maintaining these essential, proven elements and at the same time improving the fighting comfort and thereby the efficiency of our tank crews.

More and more, we are recognizing the necessity of co-ordinating the mechanical function of tanks to a degree where the least possible physical effort will be necessary to operate them. Relief of physical exertion allows the tank crew to concentrate its entire effort upon the aiming, firing, and controlling of the tank without being distracted by muscular effort.

To meet the physical handicaps and hazards of tank warfare, the Medical Department has presented to the Ordnance Development Branch its problems so that their solution can be considered in the designing of future tanks. Experience has been the basis for lessons learned, and correction as a preventive measure is the ideal solution. Prime requisites for the adoption of new improvements in tanks must, of necessity, be tempered with practical application. The whole problem is filled with complex difficulties. However, medically speaking, many suggestions are submitted to the Ordnance Department for their possible solution.

Fire from the ignition of gasoline and fire from the explosion of ammunition fall into two separate categories but they both produce the same medical result, namely, burns. Control of this hazard is obviously an Ordnance problem but the solution will parallel conditions already solved in many of our other instruments of war. Practically, the crew, in the presence of fire, must obey the impulse of "put out or get out." Ships are equipped with electric flame detectors and cylinders of CO_2 to control ordinary fires. Although ineffective against ammunition, this principle may be applied to tanks to lessen the fire hazard. Tank burns during the British campaign of the Middle East accounted for approximately one-third of their tank casualties. Burns, as a result of tank fire, still remain one of our great hazards. Preventive measures to lessen the burn hazard is a priority demand.

Fumes, gas, smoke, and dust within the crew compartment are definite menaces. Particularly annoying to any tank crew are the fumes produced by the machine guns and the large guns whose breach blocks are located within the tank compartment. At the present time, ventilation of our tanks is controlled by the fan cooling the engine which draws air into and through the fighting compartment. Tanks could be air-conditioned like any other compartment but the bulky weight of such an apparatus precludes its practical application. Proper ventilation with protection filters to exclude gas and dust will answer this problem. Here again, with controlled ventilation, temperature tolerance is essential to combat fatigue. This situation varies with the weather, time of year, and the location of the fighting. However, operational fatigue is affected by temperature and its control is of extreme importance to maintain the fighting efficiency of the tank crew.

In time of war, under stress of gunfire when a tank is disabled, it is essential that the crew evacuate the tank at once. To them, therefore, falls the immediate responsibility of getting out, removing their wounded, and seeking ground protection. Statistics have shown that tank crews evacuate 90 per cent of their own wounded. Rapid evacuation can be materially aided by increasing the facilities for able as well as disabled men. Entrances and exits of tanks insure rapid evacuation if properly placed at convenient locations to the crew. In placing these openings, due consideration must be given to the protection of the tank from gunfire, high explosives, land mines, incendiary, and chemical agents of all types. Experience has shown that these exits must be placed in horizontal planes, on top, and in the floor of the tank. Side doors are definitely a weakness and have been discarded.

Noise within the tank is still a distracting feature. It is said that tank crews become accustomed to noise but every conceivable precaution should be instituted to eliminate as much of it as possible. Crash pads of felt or hair with their comfort and safety valves distributed throughout the tank are helpful in absorbing some noise but they occupy space and can be used only where absolutely necessary. However, as a stabilizing influence, hip pads with belt attachments fastened to the seat should be encouraged in all tanks to fix men securely in their positions and permit of muscle relaxation. Racket within the tank is a technical problem which warrants continued study. This situation was brought forcibly to my attention by the drivers who test all tanks leaving the assembly line. They all complain of temporary deafness. These men, working on a daily eight-hour shift, are subject to the constant clamor of tanks undergoing shakedown and test runs over an obstacle course. They, perhaps more than the fighting forces, are subject to tank racket, day in and day out.

Visibility for tank crews is imperative to insure efficiency and confidence. Indirect periscopic vision for close-in fighting is now supplied and is efficient and satisfactory. Ports in the top of the tank for telescopic or direct vision are essential for distant firing. Their possible installation deserves consideration. Present turret tops can be opened for emergency purposes when the demand is great enough to risk direct exposure. Tanks buttoned up may be subject to blind spots, referred to as dead areas, or dead spaces. Elimination of any blind spot by complete vision in all directions at any time tends to boost the morale of the tank crew. There is no such thing as "blind driving" in tank warfare.

Operational fatigue must be held to the lowest possible level. Anything mental or physical that increases this hazard must be minimized. So many factors may enter into this problem that it is impossible to discuss them all. Suffice to say, over long periods of tank fighting, the comfort, security and morale of the crew is of first importance to secure efficiency of the highest order. Control of the temperature and humidity within the tank, protection from dust, smoke, fumes, and gas, adequate head room with easy positioning of the body with relation to the controls and firing equipment, are essential. Mechanical aids, where possible, must save excessive muscular efforts demanded of the driver, the loader, or trainer. Security of mind, confidence in the superiority of their tank, combined with a concentrated uninhibited fighting mental attitude, free from mental or physical fatigue in spite of prolonged pressure, such is the goal we are seeking and obtaining for our tank crews. In a personal letter to me, Lt. Colonel Joseph M. Colby, Chief of the Development Branch of the Tank-Automotive Center, has clarified one phase of our medical approach to tank warfare when he states: "I wish to emphasize most strongly the psychological effect upon the morale and physical condition of the crew when they have confidence in their vehicle. A tank crew with the knowledge that their armor protection, fire power and maneuverability are not surpassed by any vehicle of equal weight, and all this with greater reliability, have an incalculable advantage over another crew without this confidence in their vehicle. I have seen crews of foreign tanks physically exhausted because of worry and nervous tension, because they knew they were going into battle with odds against them."

Prompt evacuation of the wounded is just as essential in the Armored Service as it is in the marching forces. Every conceivable effort to prevent evacuation lag must be overcome. An Armored Division must never be caught immobilized but always be prepared to avacuate its wounded in order to carry on a continuing movement. Armored Medical Units now serve the Armored Forces. In tank warfare these units must be prepared to expect fractures of the lower extremities from blasting effects of anti-tank weapons, wounds from penetrating projectiles, minor injuries from splattering fragments inside of tanks, and burns. These types of casualties will constitute a large percentage of the medical work. To further meet this problem, auxiliary surgical groups are provided in the various surgical specialties, to whom these wounded men are sent.

The tank crew, under command of a Tank Commander, is always faced with the possibility of having to evacuate the tank and remove any possible casualties that may have occured. The decision of when and how to evacuate the tank rests with its Commander. On him rests the decision as to whether or not wounded can be removed before or after first aid treatment is rendered. If wounded are to be removed immediately, without first aid treatment, he can then resort to the use of morphine and, by the inverse method, remove his wounded from the tank without delay. If, however, time permits of first aid approach, pain can be controlled, hemorrhage stopped, and fractures splinted before the wounded are removed from the tank.

The British, in the early part of the war, developed rather unique methods of evacuating their wounded. The Medical Division of the Armored Forces at Fort Knox have modified, to a large degree, the British methods of removing their wounded from tanks. To this end a great deal of importance has been placed upon the use of equipment already supplied tank crews. Modification and use of the web pistol belt has answered the purpose of evacuating the wounded when time permits of such operation. A single web belt slipped from the abdomen up under the axillae, can be used as a sling. Three pistol belts in turn can be used by placing two of these belts over the shoulders and attaching them to the belt going around the abdomen and, lastly, five belts can be so arranged as to go over the shoulder and under the crotch to make a secure fastening whereby men can be removed from tanks. Other more or less arbitrary methods have been described whereby the corpsman with his more elaborate suspender sling can incorporate this equipment in such a way as to remove a wounded man from a tank with relative security. All of these plans, so carefully worked out, may, of course, be changed in keeping with possible newer developments of tanks as they come off the assembly line. Suffice to say that any effort to make a special contraption to be worn by the tank crewmen or to be maintained in the tank, has been discarded as not being practical. All efforts now are exerted toward the use of material already supplied and worn by the tank crew.

Nearly a year has elapsed since American Forces entered the African Campaign, operating for the first time in history, their own tanks. Lessons learned from practical experience and largely developed by the Armored Forces at Fort Knox, have established principles that are now the foundation for the technical procedures of tank warfare. The medical approach for the Armored Service involves the use of armored cars with half-tracks or wheel vehicles. This service, peculiar to the Armored Division, depends upon mobility and flexibility for its efficiency. For its contacts and communication, it is solely and wholly dependent upon the radio. In no other service is the radio of greater value than in this rapidly moving, flexible unit. Mobile Medical service has produced a larger field of execution in keeping with present day warfare tactics. In spite of this, the basic principles already laid down by the Medical Department, hold true although it must be admitted that officers and men attached to the Armored Division of the Medical Department must initiate self-reliance and an independence necessary in the execution of their ofttimes isolated task.

The understanding appreciation and acceptance by the Ordnance Development Branch of medical problems has led to a complete, competent, and commendable improvement in American tank warfare. Our tanks, today, present a combat vehicle adapted to an environment best suited to the fighting efficiency of its crew.

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THE SPEAKER: *It now becomes my pleasure to introduce to you Dr. Henry G. Langworthy, Treasurer and Director of the Foundation Fund.*

DR. HENRY G. LANGWORTHY: Eighteen years ago, as many of you will recall, the late Dr. Joseph Schneider, of Milwaukee, Wisconsin, left this Association a sum of money to administer along the lines of research in relation to constitutional diseases in the eye. In presenting this broad subject over the years, many noted ophthalmologists have crossed our rostrum. This afternoon, to deliver this message, I take pleasure as Director of the Fund, in introducing to you Dr. Edmund B. Spaeth, Professor of Ophthalmology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pennsylvania, who will address you on the subject, "Removal of Metallic Foreign Bodies from the Eyeball and from the Orbit."

Removal of Metallic Foreign Bodies from the Eyeball and from the Orbit

By EDMUND B. SPAETH, M. D.
Professor of Ophthalmology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pennsylvania

THE SCHNEIDER FOUNDATION EYE PRESENTATION

In this presentation on the subject, as big as it is, I am afraid I shall have to limit myself rather definitely to didactic statements, so if you will forgive me for that we will try to cover the situation.

The first point in any case under investigation is to determine the presence of a foreign body, not knowing whether it is present or not; and second, what surgical procedure are we to use for its removal. That presupposes that the foreign body is perhaps intraocular, extraocular, or a third situation which is not at all uncommon; one is in doubt whether the foreign body is intraocular or extraocular.

First is a clinical examination of the case for signs of penetration of a foreign body through the cornea, or through the sclera. The tension of the eyeball must be taken because if the eye has been opened by the penetration of a foreign body its normal tension will be lost. Other examinations would be a careful inspection to determine damage to the iris or to the lens. The presence of blood in the anterior chamber is not necessarily a sign of penetration of a foreign body. Actually, such haemorrhages probably occur more commonly in simple impact injuries than with penetration injuries.

The degree of vision which is still present is important. It is rather absurd to attempt to save a badly damaged eye from a penetrating foreign body when all vision had been lost.

Injuries to the iris and to the ciliary body. Wounds at this position, and involving these structures are potentially cases of sympathetic ophthalmia. It is far better to enucleate nine eyes badly damaged and with poor vision to no vision, than to attempt to save one eye and have thereby a totally blind patient as a result of sympathetic ophthalmia. Sympathetic ophthalmia is still the bête noir of ophthalmology. Your patient is not functionally incapacitated with one eye. Attempting to save one eye, however, unwisely may result in bilateral blindness as a result of sympathetic ophthalmia. When in doubt it is a wise rule to enucleate rather than to "take a chance."

The second problem is, if a foreign body is present what is its probable structure or alloy? Foreign bodies in the eye and about the eye in the orbit do not behave and react similarly. For instance, a sliver of glass in the vitreous may stay there for the life of that patient and do no harm whatsoever. Some foreign bodies become encysted and limit their nefarious activities in that way. A thorn, (a bit of organic material), will become encysted and as long as there is no infection carried into the eye with it there is no reason for removing it. On the other hand, such foreign bodies as any alloy or copper, regardless of size, must be extracted. Steel, especially the steel of a high carbon alloy, slowly undergoes solution in the ocular tissues and blindness may occur from siderosis bulbi.

A careful history of the situation present at the time of the accident will at times be the difference between a saved eye and a lost eye. One must go into great detail in this history to determine the direction from which the foreign body traveled; the work which was being done at the time, the type of lathe or milling machine which was being used; in fact, even the immediate emergency dressing and treatment applied are frequently most significant.

We have determined that a foreign body is present but we do not know whether it is intraocular or extraocular as yet. How are we going to handle that? We have a group of possibilities from the therapeutic standpoint.

Magnetic probes. I will dismiss them rather casually. They are to be used only during the stress and strain of such situations as battle casualties. They have a very definite place in operative ophthalmology, but they can apply only to the diagnosis and removal of foreign bodies which are magnetic.

The others, however, are for more important because they give us time for deliberation. The triangulation system of the Sweet localizer is one. Any roentgenologist can do this; as a matter of fact, in these days of master charts with these triangulation localizers it is only necessary for the roentgenologist to center the machine, take adequate pictures, and then read off his charts the localization and position of that foreign body within 1, 1.5 to 2 millimeters. The important point is to determine whether the foreign body is intra-ocular or extra-ocular, and its approximate position in relation to important anatomic elements. Naturally this leeway for errors can apply only to magnetic foreign bodies. The localization of non-magnetic foreign bodies demands the greatest exactness on the part of the roentgenologist which he can put forth.

Stereoplates and angled plates. Here a rather interesting situation exists with roentgenologists. Some swear by one and swear at the other, and vice versa. They are both wrong and they are both right as long as the ophthalmologist himself decides whether angled plates or stereoscopic plates are best for him in the case.

The other two means available are the injection of air in Tenon's capsule for those border line cases wherein one cannot be certain whether the foreign body is either intra-ocular or extra-ocular. The last is the use of radiopaque landmarks which when photographed with the X-ray plates give known fixed points for subsequent measurements. Such appliances are various phantom shells, contact glasses with radiopaque markers and the use of scleral rings sewn to the globe temporarily but at certain desired positions.

(Slide) First, the use of stereoplates. There is no doubt that in such an instance, which was one of a large metallic foreign body lying within the orbit in the outer wall of the orbit, or in the temporal fossa, that stereoplates are most advantageous for localization.

(Slide) A second situation, however, is one where angled plates were absolutely necessary. This is a case of a hunting accident. Incidentally, this case was also a medico-legal one and the angled plates illustrated beautifully the line of fire at the time of the hunting accident. In this instance, the foreign bodies, as they were buried in the orbit, in the muscles of the temporal fossa and impacted against the bones of the skull, were carefully lettered and checked one against the other on widely angled plates. The apparent rotation of the various shot, as they changed their positions, made it quite possible to decide which had become deformed by impact against the bone and were lying in the scalp. Others, through this same apparent rotation, placed themselves in the temporal fossa, inside and outside the bony orbit, and one was able to say quite definitely that they were intra-orbital but extra-ocular.

(Slide) Assuming that a foreign body lies intra-orbital, but you are in doubt as to its intra-ocular situation, the simple study of parallax will help decidedly in determining whether that foreign body lies within or without the globe. In such a situation X-ray photographs are taken with eyeball directed straight to the front, rotated 45° upward and 45° downward. The position of the foreign body in its relationship to the center of rotation of the eyeball will control the degree of displacement of the foreign body based upon this 45° angle rotation. A simple line drawing can be made, and from it one should determine if the degree of displacement of a foreign body compares exactly with the degree of displacement the graph should indicate. Failing this, one may be certain that the foreign body lies extra-ocularly, even though some displacement may be present as the result of movement of muscles in which the foreign body may be buried.

The use of radiopaque landmarks. (Slide) Here is the case of a young lady with a very definite fistula opening into her internal canthal angle, with a foreign body at the base of that fistula. There was no way of telling definitely the position of that foreign body, whether it was intra- or extra-orbital, intracranial or intranasal, by triangulation method, by stereoscopic plates, but one can lay out radiopaque landmarks and thereby determine the localization of the foreign body exactly.

(Slide) The fistula was injected and the posterior limits of the lipiodol injection seen laterally and posteriorly. Combining the two, a radiopaque cage over the cornea and corneoscleral shoulder with the lipiodol injection and a lateral view, and one could see that the foreign body is localized as lying extra-orbital and intracranial. It was removed uneventfully thereby. It was, incidentally, a non-magnetic foreign body.

As to the handling of magnetic and non-magnetic intra-ocular foreign bodies, first a few words as to magnets. Two major magnets are used in ophthalmology, one a giant magnet and the other a hand magnet. They are in no way interchangeable in use one with the other. The giant magnet is not only a therapeutic instrument but it is also a diagnostic instrument. A good giant magnet must be sufficiently strong to pull a tiny steel ball with a force of over thirty times its weight at a distance of 20 millimeters from the magnet tip. The only absolute essential is that the foreign body be intra-ocular and behind the iris and ciliary body diaphragm. The value of the giant magnet lies not in its increased pull but in its proportionately increased width of magnetic field; hence the degree of pull can be even further controlled effectively by varying the distance between the foreign body and the tip of the magnet. In British literature this has been demonstrated beautifully rather recently in their handling of intra-ocular magnetic foreign bodies as a result of battle injuries.

The giant magnet is not to be used at any time for extracting a foreign body from the anterior chamber of the eye.

The hand magnet. As the giant magnet has its greatest value in a demand for a diffuse magnetic field which permits, in fact demands, variability, so the hand magnet has its indications in cases in which the magnet tip can be applied directly or approximately to the foreign body at an interval of no more than 2 millimeters.

To recapitulate, a giant magnet is for use when one can approach within 20 millimeters of the foreign body; the hand magnet when one can either touch it or approximate it with interposing tissue only 1 or 2 millimeters distant.

Lancaster's rule for a good hand magnet is that it must pull a tiny steel ball in contact with its tip with a force of several thousand times its weight. To emphasize, a giant magnet and a hand magnet are not interchangeable in use.

There is a tremendous amount of controversy among ophthalmologists as to the removal of a foreign body through the posterior route and through the anterior route. Shall the foreign body be taken out through the scleral coat, presuming that the particle lies behind the iris-ciliary body diaphragm, or shall it be coaxed around the suspensory ligament of the lens and into the anterior chamber of the eye through the pupillary aperture and there removed by a clean incision in the anterior chamber, that is, in the cornea at the angle? There is not the slightest doubt whatsoever that the posterior route surgery results in the highest percentage of lost eyes. The anterior route surgery results in a considerably higher percentage of successful operations and successful extractions.

There are certain situations, however, which must be considered wherein the posterior route may be used or even must be used, or reversely, where the anterior route may be used or must be used. In general, the smaller the particle the more is the latter, that is the anterior route, indicated; the larger the particle the more is the former, that is the posterior route, permitted. The wording is deliberate. In my opinion, the posterior route is never an optional situation or choice with magnetic extractions unless with very large fragments, with large entrance wounds of penetration, and when large fragments have traversed the eye and lie in a position where a posterior sclerotomy will be away from the vortex veins behind the equator of the eyeball and where damage might occur to the lens during an anterior route extraction. An intact lens should be left so. Hence, a damaged lens as the result of the penetration of a foreign body offers no contraindication to anterior route extraction except for large foreign bodies.

The do's and do not's of posterior route extraction are rather definite. One must stay behind the equator with the sclerotomy. One must avoid the large scleral vortex veins if any vision is to remain after surgery, because, in the final analysis, the removal of a foreign body presupposes usuable vision following the extraction of a foreign body. The sclera must be sutured after the operation.

The matter of retinal detachment following posterior route extraction is a most serious subject. There is no doubt whatsoever that this complication follows entirely too commonly after this surgical procedure. On the other hand, if not already present, it is doubtful whether an anterior route extraction presupposes to this postoperative complication. Magnet tips should never be introduced into the eye. It is utterly hopeless to attempt to remove an intra-ocular foreign body by introducing an instrument into the eye, and then magnetizing this instrument by contact with the head of the magnet. It is simply "working in the dark" and irrevocable damage is certain to result.

The do's and do not's of the anterior route extraction. It is a very interesting procedure to watch a capable man extract a foreign body from the eye by the anterior route. There is nothing more beautiful in surgery when that is carried through well. The magnet tip is to be started as far posteriorly as is possible. Then, with the magnetic field closed, the foreign body will pass to that magnetic field by its shortest possible route. Then, as the magnet is carried slowly forward, the foreign body should pass through the suspensory ligament of the lens into the posterior chamber of the eye. From there, it can be coaxed around the iris on its posterior surface, through the pupillary aperture and into the angle. It is now visually present, and it is a very simple matter to pick up a keratome, to make an incision at the anterior chamber angle, and with a hand magnet lift the foreign body out of the eye.

If, in carrying the foreign body through the angle of the iris and the ciliary body, it becomes incarcerated there, discontinue any further magnet attraction. An iridectomy should be made at the angle immediately, and the foreign body extracted through this operative coloboma.

Non-magnetic intra-ocular foreign bodies. There are three procedures available here and these also are not interchangeable one with another. Each is not an operation of desire but an operation of necessity. Each has certain optimum indication. First, the use of radiopaque landmarks. This will permit the formation of a flap sclerotomy and extraction. Second, the use of an endoscope, which is not greatly unlike a cystoscope or a bronchoscope. Third, biplane fluoroscopy.

(Slide) This is a case of a foreign body, a scale of copper which lay at a certain position. It was accurately localized. Two radiopaque landmarks were sutured to the sclera—both silver rings of a known size. Subsequently X-rays were taken for the correction of their position; the second ring, the smaller of the two by measurement, sutured immediately over the site of localization. A flap sclerotomy was made inside of that, and the foreign body picked out of the choroid without damage whatsoever to the eye. This procedure would not have been possible without the exact localization possible by such radiopaque landmarks.

(Slide) The use of an endoscope. With the use of an endoscope, one must have a clear vitreous. The endoscope is passed through the scleral incision, and by the use of various shaped forceps through the telescopic tube, one can grasp the foreign body and remove it. Forceps are arranged for various shaped foreign bodies. Those shown in this slide are for a BB shot, another one is for a scale of copper, and another for the removal of a sliver of glass.

Third, the use of biplane fluoroscopy. Biplane fluoroscopy, there is no doubt, has its optimum indication in the removal of extra-ocular foreign bodies and in those situations wherein the foreign body must be removed. In the presence, however, of an opaque vitreous one cannot use the endoscope, hence the biplane fluoroscopy technique is the operation of necessity. It is rather unfortunate, however, that there is considerable trauma connected with the use of the biplane fluoroscope and with the grasping forceps, and while the incidence of removal of foreign bodies is high, the incidence of satisfactory visual acuity following the use of the biplane fluoroscope is not especially gratifying.

To abstract this rather brief presentation of certain salient situations, there are six points I should like to call to your attention.

1. The great and important possibilities of roentgenograms, frequently not used adequately, both flat, angle views and stereoscopic views. These are problems not alone for ophthalmologists; these are problems for every physician.
2. The use of air injections into Tenon's capsule for more accurate localization of extra-ocular particles.
3. The use of radiopaque appliances for localization, but especially as an aid to the extraction of non-magnetic foreign bodies by means of a flat sclerotomy.
4. The use and indications and dangers of the biplane fluoroscope.
5. A consideration of certain important factors in the use of magnets, both diagnostic as well as therapeutic.
6. Indications for and the use of an endoscope, which is a sound surgical instrument and has been successful in at least sixty per cent of instances wherein it has been used.

Artificial Insemination in Sterility

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I think perhaps it might be fitting if we turned the lights low and played the New World Symphony, because I am going to talk about test-tube babies; at least that is what the lay people would have us believe—a term which, of course, we all abhor. The technic of artificial insemination has caught the lay fancy and there is more drivel and rot published about it than almost anything in the whole gamut of medicine.

How old is artificial insemination? Is it a brand-new technic? I am afraid I cannot answer that categorically. All I can say is that there is a rather pleasant legend which goes back to the fourteenth century that artificial insemination was first used by an Arab sheik. When making war upon a neighboring tribe his men stole into the enemy's camp and artificially inseminated the fine-bred Arabian mares with semen from an old broken-down stallion. That is hardly a technic that we can use in modern tank warfare, but it is the only way I can tie up this talk with the war effort, which seems to be the program for the evening.

The first scientist to work with artificial insemination was the very interesting anatomist, Lazarro Spallanzani, who, as his name suggests, was an Italian. In 1780 he thought up the experiment of artificially inseminating a dog. He chose a brown and white spaniel, closeted the bitch in his room for thirteen days until she went into heat and then he got eighteen grains of seed from a male dog, which was black in color, and inseminated immediately the bitch with the seed from the male dog. He kept her in his quarters for twenty more days, which shows a great tolerance for dogs on the part of Spallanzani. He then let her loose and sixty-three days after she was inseminated she gave birth to pups which resembled the sire.

The first human case was done by the famous philosopher-surgeon-physiologist, John Hunter. His case was apparently done around 1790, but not published until 1799. A couple consulted Hunter. The man had a hypospadias. Hunter advised the husband to collect his semen and to inject it into his wife. This was done. She became pregnant and gave birth; as far as we know this is the first human case of artificial insemination.

The next milestone in the story is J. Marion Sims, the founder of the Woman's Hospital in New York City, the inventor of the duckbill speculum, the repairer of vaginal fistulae, the man who operated with equal success on crossed eyes and clubfeet,—a very remarkable surgeon. Marion Sims experimented with artificial insemination and inseminated six women, a total of fifty-five times and got one take; but unfortunately this patient aborted, according to Dr. Sims, from a fright.

Following this experience little was done with the technic until the Russian veterinary physiologist, Ivanoff, in 1899 began to use artificial insemination in animal husbandry. He published a very remarkable monograph in 1907 and since then, as you probably know, Russia has made tremendous advances in the field of artificial insemination as applied to animal husbandry. The Soviet government has animal breeding stations scattered throughout the great nation and each peasant is privileged to bring his mare or his cow or his sow or his ewe to this station and she is inseminated from the semen of a well-bred male of the same species.

According to Kersin, in 1936 there were 650,000 horses born in Russia as the result of artificial insemination. The remarkable thing is that one good male can inseminate a large group of females; Kersin reports that one ram inseminated 15,000 ewes in 1936, and one bull 1,000 cows. The proportion of conception in the ewes was 96 per cent and in the cows was 93 per cent.

We physicians to the human race are leagues behind in artificial insemination, trammeled as we are by conventions, moral codes and frailties of character which never hinder the stock breeder. Since Engelmann published a paper in 1928 reporting 165 human cases collected from the world literature, there have been many papers and many cases added, so that I dare say that there are now several hundred or even a thousand human cases.

What are the indications for artificial insemination? Leon Israel of Philadelphia divides artificial insemination into two groups: the so-called homologous, in which you use the semen of the husband to inseminate his wife; and the heterologous, in which you use the semen of an unrelated and fertile donor to inseminate the patient.

Homologous insemination, in which we use the husband, has two large indications: (1) that group in which intravaginal coitus between two fertile individuals is mechanically impossible — impotence, hypospadias, vaginismus, tumors, obesity, and so forth; (2) a large group in which the husband's semen is injected to cure a chronic sterility for which we can find no cause, or the cause is a subnormal semen. In this group, which is a large wastebasket, if the patient does not get pregnant after three or four years and the doctor has put the man and woman through complete studies with zero results, then he is prone to resort to artificial insemination—a very bad practice from my point of view. In my own experience it has yielded absolutely nothing.

The third group is the group in which we do the heterologous artificial insemination, because we have a sterile husband or a husband who has such a bad heredity that it would be dangerous for him to sire his own child.

I just want to say a word, in order to dismiss it, about the technic of testicular aspiration. There are some physicians who recommend the aspiration of the testicle when the vas deferens is sealed off. The spermatozoa are aspirated from the testicle, put into Ringer's or Locke's solution and then injected into the uterus. There has been only one successful case in the history of medicine as far as I can learn. This case was published by Mr. Wilson, an English surgeon, in 1929. There is no other case in the history of medicine in which testicular aspiration from a man with a blocked vas has succeeded in impregnating his wife; obviously such poor odds make it a useless procedure.

On whom should we do artificial inseminations? It is obvious that the couple in whom coitus is physically impossible presents an excellent indication for artificial insemination. The semen is collected, injected intravaginally, and the results are usually very satisfactory. The second group, the chronically sterile, the husband with the subnormal semen, in my humble estimation in no way offers an indication for artificial insemination. I know it has become the vogue of the doctors throughout the East and I think probably throughout the country to say to a patient after they have worked on the case for months and months, "Well, now we will finally do the one thing left and that is artificial insemination." I have done that in eight couples. I succeeded in none, but in two of these couples the women later became pregnant per via naturalis. I do not think that artificial insemination in a woman who is normally formed and normally built and in a couple who can have normal intercourse has any possible advantage over normal intercourse, and I decry and deplore the widespread medical policy and procedure of doing artificial insemination in these cases.

In what percentage of our sterile couples should a heterologous insemination be done from an unrelated donor? We examined 208 specimens of semen at the Johns Hopkins' Hospital from cases in whom the sterility had existed for a period of at least three years. In these 208 couples we found that the man's semen was perfectly normal in 62 per cent—obviously no point in using a foreign donor; in 16 per cent the sperm count was between 30 and 60 million, the normal count being around 90 million—borderline cases. In these I would certainly attempt to improve their fertility, especially the sperm count of the male, rather than resort to artificial insemination. In 12 per cent we found the count from 10 to 30 million; in 7 per cent less than 10 million, and in 2.5 per cent a complete absence of spermatozoa.

I feel that in the 10 per cent who either have a count chronically below 10 million or who have no spermatozoa, doctors have an honest obligation to suggest artificial insemination from a foreign donor. I would work with the borderline groups for months or even years first, and then if no success, perhaps suggest artificial insemination if the husband's count is perpetually below 60 million.

Technic. How do we select our donors for this heterologous technic? I am fortunately located, because we have a large medical school and we simply buy the semen specimens from either interns or from young house officers. We pay a relatively low price for it. We buy it just as one buys blood for transfusion. We get specimens from five to fifteen dollars, depending upon the affluence of our recipient. The donor simply masturbates the specimen or practices coitus interruptus and collects the specimen in a large glass vessel, transfers it to a small bottle, and then it is brought to my office.

We do not know how long a specimen will keep, but several of my successful cases have been done with semen at least two hours old, and if you have to keep the semen for any length of time it should be kept at 6 degrees Centigrade, because at room temperature of course it deteriorates rapidly. There have been cases of artificial insemination in animals in which the semen has been collected at Beltsville, Maryland, and an artificial insemination done down in the Argentine, taking seven days to fly the semen down, and a take has occurred. Thus in animals we know that a long latent period may exist. We do not know this about the human.

The method of injection I think should be very simple. All I do is simply expose the cervix with a bivalve speculum and then I take a Luer syringe to which I attach a metal intravenous cannula and simply put the cannula just within the external os, not tight against the os but just inside of it. I then put my finger on the plunger of my hypodermic syringe and the 2 or 3 cc. of semen are spurted into and at the external os by three or four thrusts of the plunger. Meanwhile, the patient's hips have been elevated. She remains in this elevated position on her back for twenty minutes. I put a small piece of cotton at the introitus to keep her from soiling her clothes and then she can get up and go about her day's routine.

We are experimenting with two modifications of this technic to increase our chances for success. As you know, Hotchkiss and McLeod have published a very interesting article in a current number of the American Journal of Obstetrics and Gynecology on a fortified Ringer's solution which is injected precoitally in sterile couples for whom they can find no reason, and from such a simple procedure got twelve pregnancies. The medicament is put out under the trade name of Nutrisal. I have been using Nutrisal simply to lavage out the vagina before I inject the semen to give the spermatozoa probably a more perfect environment.

Dr. Schwenk and Tislowitz, of Metz, have worked with a substance called the spreading factor, which is a testicular enzyme which will dissolve mucus, and I am now working also on that and applying it to the cervix to dissolve the mucus before using artificial insemination. I cannot tell you what these two modifications of technic are worth yet.

I choose the eleventh and fifteenth days of a 28-day cycle for my first insemination. If a patient has, say, a 31-day cycle I simply add three to my eleven and three to my fifteen and use, then, the fourteenth and eighteenth days. If she has a 26-day cycle I simply subtract two and use the ninth and thirteenth days. If I do not succeed the first month, then I go forward one day or go back a day and in that way simply alter the days by one or two each month until I have well covered the fertile period.

My results are not extraordinary. I have had two cases in the Group A, in which coitus was impossible, in which the husband was impotent and could simply masturbate a specimen. This was done and the specimen brought from his home to the office. We inseminated the two wives, and in both instances we got a take, so I am batting exactly 100 per cent in these two cases, which is hardly a series.

I have tried eight in which the woman appears normal and the husband appears normal or he has a subnormal semen and I have never had a positive result. In this series of chronic sterility my average is triple zero. I have tried forty-nine cases in which I have used a foreign donor, heterologous insemination. Of these forty-nine cases I have had twenty-one pregnancies. Six patients are still under treatment or were treated this month and therefore I believe I can leave them out of my results, so I can say I have had twenty-one pregnancies in forty-three cases. Eight of these forty-three cases only had three or four inseminations—hardly a fair trial. The other fourteen patients had a fair trial and I can say we failed. So we have had twenty-one pregnancies in thirty-five cases in which there has been an honest effort. These results are not remarkable, but after all you can't expect any better result than you get from normal coitus. After all, if you had thirty-five supposedly fertile women attempting pregnancy through the normal means, probably only twenty-one of them would succeed in a matter of the several months in which we do an artificial insemination, and remember we usually do only two inseminations per month. So I think you can expect about 66 per cent success if your patient will persist for six or eight months, if it is necessary, to get the result which you seek.

The interesting thing in my work is that seven of my patients got pregnant the first month, eight got pregnant the second month, so of my twenty-one successes, fifteen occurred in the first two months' try, and from that I may say that if you don't succeed immediately, within the first two months, then the prognosis is not so very good. Three succeeded the third month, one the fourth month, three the fifth month, and one the thirteenth month. This woman is very interesting because she had a retroposed uterus. I did a Rubin's test on her and found that it required about 140 millimeters of pressure to get the air through. I finally persuaded Dr. Telinde, the gynecologist, to suspend her uterus. The air then went through at 60 millimeters of mercury pressure and she was impregnated by artificial insemination the second month after we had suspended her uterus, so I suppose in her, which is a rare finding, this retroposition with pressure on the tubes actually was a strong sterility factor.

There are moral and legal problems involved in heterologous or donor artificial inseminations. I have certain ideas about these and I have established for myself six rules.

1. The donor must remain completely anonymous to the recipient and the husband, and the recipient

and the husband must remain equally anonymous to the donor.

2. Before attempting artificial insemination know the couple, their intellectual capacity and emotional stability, and, if possible, the likelihood of a permanent marriage. Only a small percentage of patients applying qualify for so radical a social procedure. When a doctor consents to do an artificial insemination from an unrelated donor it is really the couple's insignia of good character. Artificial insemination must always be completely individualized. It should never be an assembly line kind of medical treatment.

3. Never urge the procedure. If either husband or wife is lukewarm, drop it completely.

4. Forget signed papers. If the patients are carefully selected, contracts and agreements are unnecessary and simply act as a permanent reminder of something which should be forgotten as quickly and completely as possible. In the ideal case, by the time the patient reaches term, the woman, the husband and the doctor have to think twice to remember that the pregnancy is physically not the husband's, for psychically it has become his.

5. If you deliver the patient and know the facts—and I don't see why you shouldn't deliver her—falsify the hospital record and the birth certificate. Here a white lie is a kindly humane act.

6. Keep the fees low. Keep artificial insemination out of the mercenary column. It is no way to get rich quick. View it as a personal medical service, the contribution of an humble Aesculapiad to the happiness of an unhappy couple.

Injuries to the Head in Time of War*

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The severity of war wounds of the head has been greatly reduced by protective helmets and, the mortality has been lowered by the use of the sulfonamides, locally and orally, blood and blood plasma intravenously, and greater facilities for transportation of the wounded.

Since the war began a great deal has been written concerning the treatment of head injuries sustained in the field, aboard ship, and during air raids. Air raid casualties among civilian population make it just as imperative for every civilian physician to know the essentials of the early care of head injuries as the members of the medical corps of the Army and Navy. During air raids it will be necessary for all physicians to render first aid treatment, select the cases in which immediate surgery is imperative, and decide which cases are beyond medical care. In no other group of injuries is this so important as in injuries to the central nervous system.

We are often asked if it is possible to classify war injuries of the head, both from the diagnostic and the therapeutic standpoint. This can be done only in a general way, since many factors are involved, such as associated injuries and the general condition of the patient. A patient may suffer a mild head injury and a chest injury, a severe burn, or a fracture of one or more extremities. The treatment should be directed toward the more severe of the multiple lesions, and a compromise in management may be necessary.

We have been given a simple classification by Cairns based upon his experiences with head injuries in Great Britain.

A. Closed head injuries; cases in which the scalp is unbroken.

B. Open head injuries; cases in which the scalp has been opened.

1. Scalp wounds without fractures of subjacent skull.
2. Compound fractures of the skull, fissured or depressed.
3. Penetrating wounds of the skull from projectiles.

The closed group of injuries includes any injury to the head in which there is no evidence of break in the skin. They range in severity from mild concussion to massive fractures with underlying damage to the brain. They differ from the open type of injury in that they are not complicated by infection except when rhinorrhea develops from a fracture through the cribiform plate or the fracture involves one or more of the sinuses. In this group are the cases in which surgery is rarely urgent and first aid treatment and observation are important because intradural and extradural hemorrhage may become evident.

In the diagnosis of this type of injury the state of consciousness as well as the pulse rate, blood pressure, and respirations are more important than the evidence of fracture. The unconscious patient may have suffered a mild concussion and the recovery may be immediate; or he may have sustained a severe damage to the brain and recovery may be delayed. All unconscious patients should be cared for immediately and if their condition is not too serious they should be transported to first aid stations or hospitals for observation and treatment.

The term concussion has long been used in connection with the unconscious patient who has sustained a closed head injury. The exact cause of the loss of consciousness remains unknown but the causes to which it has been attributed are anoxia and edema of the brain, physical and chemical changes in the cells, and the physical effect of acceleration and deceleration of the head. Regardless of the cause a patient with true concussion will recover with conservative treatment consisting of supportive measures, rest, and adequate use of oxygen. Most of the patients who are unconscious as a result of a closed head injury are allowed to lie flat on their back until treatment is decided upon and may even be transported in the same position. The jaw is relaxed, the tongue is swallowed, and the patient breathes with difficulty and often becomes cyanotic. Not infrequently have I been called to see such a patient who is suspected of having Cheyne-Stokes respiration only to find that he is strangling on his swallowed tongue. These patients should be turned on their side, other injuries permitting, or even on their abdomen with one shoulder elevated and the head turned to one side. This insures easy breathing and the escape of saliva from the mouth. Most of the large emergency hospitals have instituted this position in all their emergency head injuries.

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*NOTE—The opinions or assertions contained herein are the private ones of the writer, and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

Restricted breathing in these cases contributes to the anoxemia of the cerebral cells. Prolonged anoxemia of the cells is followed by irreparable damage. It has been demonstrated experimentally by Schnedorf and his associates that brain concussion with or without skull fracture causes a depression of arterial blood oxygen from 5 per cent to 44 per cent below normal so that we have a definite potential oxygen deficiency of the cerebral cells in all head trauma. Thus the first consideration of the unconscious patient is unobstructed breathing, and the administration of oxygen by means of masks or intranasal catheters has been followed by clinical improvement. How much of the so-called post-traumatic syndrome can be attributed to the lack of oxygen and the resultant anoxemia of the cells remains to be demonstrated. But the clinical and experimental evidence strongly suggests that it is an important factor.

The diagnosis of concussion, contusion, and laceration of the brain in closed head injuries can only be made after observing the patient for some time. Patients suffering from concussion regain consciousness within a short time with a few residual symptoms. Those with contusion and laceration continue in a semicomatose state or even develop a state of restlessness or mania and require extreme sedation. Morphine should never be used in head injuries. Where there are associated injuries such as painful fractures or burns, it may be necessary to compromise and administer morphine. Codeine and the barbiturates should be used, and in extreme restlessness paraldehyde can also be given.

The employment of spinal punctures may make possible a differential diagnosis. When the pressure of the cerebrospinal fluid is high and the fluid bloody, a more serious condition than concussion should be suspected. Roentgenograms may show fractures which when present indicate the extent of cerebral damage.

Consequently these patients with closed head injuries need careful observation, for immediate or delayed intracranial bleeding may develop to the point where operation is imperative, especially the bleeding from a severed middle meningeal artery. Fluids should be administered, preferably hypertonic dextrose either in 50 per cent solutions in quantities of 50 c.c. or in larger doses of 500 c.c. of the 20 per cent or the 10 per cent solutions. Magnesium sulfate enemas should be given, and should the unconsciousness last for several days an intranasal tube should be inserted and high caloric formulas be given. Sedatives can also be administered through the tube. Spinal punctures should be done when indicated.

In a discussion of open head injuries scalp wounds should be considered first. In civil practice the scalp wound is usually of small consequence, but, referring to scalp wounds in warfare, Cairns states, "The apparently trivial operation of cleaning and suturing a wound of the scalp is probably the most important neurosurgical operation of the war."

During a British symposium on casualties during air raids it was emphasized that there was nothing more dangerous than an innocent-looking, penetrating scalp wound. A physician attached to a first-aid station remarked that on a particular air-raid night he had treated a certain number of small innocent-looking wounds of the scalp. "Yes," replied the surgeon who had received the cases, "you sent me 11 in one night, four of them had foreign bodies in the brain and the other 7 were depressed fractures."

The emergency treatment of scalp lacerations consists of shaving the scalp, controlling the hemorrhage, cleaning the wound without a great deal of manipulation, introducing sulfanilamide powder, and bandaging with sterile dressings. These patients should then be transferred to a hospital or sick bay where facilities are available for extensive surgery if necessary.

Scalp wounds with fracture of the subjacent skull can frequently be diagnosed at the first-aid station. Here the extent of the injury can be appraised and the wound dressed by shaving the adjacent scalp, controlling the hemorrhage, cleansing the wound and instilling sulfanilamide powder and applying a sterile bandage. As with simple lacerations of the scalp the question of transportation of these cases and the decision of the importance of early operation rests with the physician, for with the use of the sulfonamides it has been found that closure of these wounds can be delayed in the less serious types of injuries for from 24 to 72 hours.

This allows for a selection of cases for rapid transfer and alleviates the necessity for immediate debridement and closure in all cases.

It is sometimes difficult to differentiate between innocent-looking scalp wounds and the penetrating wounds of the skull. Cushing referred to the walking wounded, those patients who had received penetrating wounds of the skull from shell fragments and yet were not disabled. The experience of the British in the present war has emphasized the necessity of suspecting all cases of scalp wounds and getting roentgenograms of the head routinely whenever possible. These patients should be treated from the emergency standpoint in much the same manner as the foregoing patients, but when definite penetration is evident they should be transferred immediately for more extensive investigation and operation.

In carrying out debridement in extensive open head injuries all damaged brain tissue should be removed at the time of operation. This can be done sometimes without enlarging the opening of the skull. When a small skull wound has been produced by a shell fragment or bullet it may be necessary either to enlarge the opening with bone biting forceps or, as the French have suggested, to reflect a bone flap containing the defect. Authorities differ with regard to the type of opening to be made, and it is of relatively little importance depending on the nature of the wound and the judgment of the operating surgeon. Jefferson points out the advantages of the triradiate incision about the opening in the scalp and the debridement through this type of opening: "Its advantages are that an adequate exposure can always be obtained, the minimum of a gap occurs if the wound breaks down, and drainage tubes can be conveniently placed."

All operations upon the head should be preceded by a complete shaving of the scalp and should be done whenever possible under local anesthesia with infiltration of 1 per cent novocaine solution or under pentothal sodium anesthesia given intravenously. Intratracheal gas or ether, or avertin by rectum may be used. Careful and complete asepsis should be maintained throughout the operation. Fragments of bone and shell should be removed as well as any particles of dirt and clothing. The damaged brain tissue should be carefully sucked from the wound. Mechanical suction if available is ideal, but a small catheter and a large bulb syringe can be used to great advantage. Bleeding vessels can be clipped with metal clips, ligated, or coagulated. When finished, the wound should be dry and no damaged brain tissue should remain. The wound should be covered with a thin film of sulfanilamide powder preferably blown into the wound by an atomizer. The dura should be closed with interrupted silk sutures whenever possible and the scalp approximated with two layers of silk, one in the pericranium and one in the skin. Sulfanilamide should be blown into the wound after the dural closure and before fixation of the bone fragments or bone flap.

Sulfanilamide has been found most suitable and efficacious for open wounds of the head. Sulfanilamide is the most soluble of all the sulfonamides, and there have been cases reported in the literature in which sulfathiazole and sulfadiazine powder used in brain wounds produced irritative symptoms evidenced by Jacksonian seizures. When these brains have subsequently been explored a small amount of unabsorbed powder in the form of a cake has been found producing the irritation.

Of all the sulfonamides for oral administration in wounds of the brain, sulfadiazine is the drug of choice at present because of its diffusion in the cerebrospinal fluid. In all open wounds of the head and especially when the ventricles have been penetrated, it should be given over a period of 7 to 10 days. Also whenever there is a leakage of cerebrospinal fluid from the ears and nose in fractures of the skull sulfadiazine should be given.

The matter of spinal punctures has been mentioned in connection with closed head wounds. It has always remained a controversial subject, but recent experimental work has pointed strongly to the fact that lumbar punctures are of diagnostic and therapeutic value and can be done with safety.

Lundquist and LeRoy have reported some interesting experimental work in which they showed that within an hour after trauma sufficient to cause contusion to the brain of dogs, there was an average decrease of 24 per cent in cerebral oxygen consumption and of 17 per cent in cerebral blood flow. Twenty-four hours later there was little change in values if untreated.

The administration of 50 per cent glucose or sucrose to dogs with a 24-hour head injury was followed within an hour by a striking increase in brain oxygen consumption, but no significant change was noted in the cerebral blood flow.

Drainage of the cerebrospinal fluid in dogs with a 24-hour head injury was followed by an even greater increase in brain oxygen consumption and by a 25 per cent increase in the brain oxygen consumption and by a 25 per cent increase in intracranial blood flow.

From these experiments it can be inferred that the administration of hypertonic fluids and drainage of cerebrospinal fluid are valuable in the protection of the patient from anoxemia of the cerebral cells.

Cerebral edema and the dehydration regimen for this condition has not been discussed. It is apparent that such a condition does occur and that adequate treatment should be instituted. However, with the clinical and experimental evidence of an associated oxygen depreciation of the cerebral blood, the decreased oxygen consumption of the cerebral cells and the lessened blood flow, the necessity for drastic dehydration seems to be of relatively little value. It is of far more importance that all patients be treated individually, and if fluids have been lost through perspiration or loss of blood they should be supplied. The use of serum albumin for the treatment of shock has proved of extreme value in dehydrating the cerebral cells. That the administration of hypertonic dextrose or sucrose is of value has been demonstrated both clinically and experimentally. The restriction of fluids immediately after the head injury has been sustained has its clinical values, but we have learned a great deal about fluid balance and the necessity for such a physiologic stabilizing mechanism. Blood transfusions are frequently necessary as well as the administration of plasma in the treatment of these cases.

The reports from the war zones have demonstrated a surprisingly lowered mortality in injuries to the head. Probably one of the most complete of these reports is that of Major Peter Ascroft which has just been published. From his two years experience in Africa he reported on 516 consecutive cases of wounds of the head due to missiles and found that 85 or 15 per cent of the patients had suffered scalp wounds, and 139 or 27 per cent fracture of the skull with an intact dura. There were no deaths in the group with scalp wounds and but two deaths in the fractured skulls with dura intact with a mortality of 1.5 per cent. There were 292 or 57 per cent of these which had sustained fracture of the skull in which the dura was pierced resulting in only 44 deaths. In the entire 516 cases there were 46 deaths or a 9 per cent mortality. In the 436 cases which recovered he found that of those who had involvement of the scalp alone, of which there were just 80 cases, there was only one invalided on account of his wound and this was due to wounds elsewhere and the rest were returned to duty. Of the 130 cases in which the wound involved the bone, 10 were invalided on account of wounds, 110 or 85 per cent were returned to duty. The dura was involved in 226 cases, 93 of whom were invalided on account of head wounds and 124 or 55 per cent were returned to duty. In a total of 436 cases, 71 per cent of them returned to duty. This is an amazing figure and unparalleled in any previous war with regard to the number of patients who had head injuries and returned to duty. He called attention to the difference between operating in the forward areas and operating at a base hospital and emphasized that incomplete operations were more dangerous than a few hours delay.

In this war we have found that operations can be carried out up to and including 72 hours after the wounds of the head have been sustained. In going over his cases Ascroft found that of the operations in the forward areas 5 per cent were operated upon within 8 hours, 27 per cent in from 9 to 16 hours, 41 per cent in from 17 to 36 hours, and 22 per cent in 36 to 72 hours and only 5 per cent were operated upon in forward areas over 72 hours. Of the evacuated cases in which the primary operations were at the base, 15 per cent were carried out between 17 and 36 hours, 48 per cent between 36 and 72 hours, and 37 per cent were operated on after more than 72 hours had elapsed. The three valid reasons for retaining head injuries in the forward areas were, severe shock, urgent surgery and no reasonable hope in reaching the base for 48 to 72 hours.

He used the sulfonamides locally and by mouth maintaining a concentration in the cerebrospinal fluid of 10 mg. per 100 c.c., even if intravenous administration was necessary. Where cases were threatened with, or actually had developed meningitis, a crebrospinal fluid level of 15 mg. per 100 c.c. was maintained.

It is apparent from the reports in the literature and the observations of the medical officers in charge of battle casualties, in which head injuries have been sustained, that the protective helmet has reduced the number and severity of the lesion. Also the emergency treatment depends upon the condition of the patient. When shock is present it should be treated. When no open wounds are found and the patient is unconscious he should be treated expectantly with proper posture for transportation, providing free breathing to prevent anoxemia and aspiration of saliva. When open wounds occur the scalp should be shaved, hemorrhage controlled, the wound cleaned, sulfanilamide inserted, and the wound covered with a sterile bandage. Cases should then be selected for rapid or delayed transportation according to the severity of the wound. No extensive surgery should be done until neurologic examination has been made, roentgenograms of the head have been taken, and facilities for extensive debridement and closure are available.

Multiple wounds may influence the treatment of head injuries and in every case the general condition of the patient is important. Nutrition and fluid balance should be maintained. Patients should be encouraged to be up and about as soon as possible and rehabilitation should be started early in the convalescence. The return to active duty of such a large percentage of head injuries sustained in battle is sufficient testimony to the improved methods of treatment and management of these cases.

Medical Department Activities on our Various Battle Fronts during the Present War

By NORMAN T. KIRK, M. D.
Major General, United States Army, The Surgeon General, Washington, D. C.

It is a great pleasure to have the opportunity to address this gathering. If I remember correctly, some twenty years ago I helped my old chief, Colonel Keller, prepare a clinic for this Association at Walter Reed Hospital where we showed you some amputation stumps and bone grafts from non-union and lost substance as a result of World War I and where Colonel Keller showed you the treatment of chronic empyema that he had developed there at Walter Reed to clear up that great group of cases that followed our influenza epidemic during the last war.

Tonight I want to speak about what your contemporaries are doing in our armed forces, as part of those armed forces, winning this war for you and me so that we may have a decent home to live in in America. They are doing a grand job. I have seen it myself. I will go into that later.

I have a letter here from General Clark, Commanding General of the 5th Army, now fighting in Italy, and if you will allow me I will read it to you. This is from the Commanding General, Headquarters 5th Army, in the Field, dated 25 September 1943.

"Dear General Kirk:

"I desire to express the highest commendation for the wonderfully fine work performed by the medical units of this Army. Their devotion to duty under the hazardous and trying circumstances of the landing at Salerno Bay and their skill and efficient administration reflects the best traditions of the Service. Many wounded officers and men who will eventually be restored to full duty would have died but for the efficient work of the Medical Corps. I am especially pleased with the performance of the Surgeon of the 5th Army; he has done a magnificent job.

"From the first landing to the date of this letter, 3,335 casualties have been admitted to the hospitals of the 5th Army. The first hospital opened within three to five miles of the front line. Another hospital began to function the following day still closer and under more difficult conditions. Neither hospital had any nurses when opened. Thus far there have been only 42 deaths in the hospitals. Thirty-two of these cases were those of U. S. personnel who died from wounds. Five were U. S. personnel who died from disease or injury; five were enemy who died of wounds. Many of those who died would never have reached the hospital alive had the hospitals been located at a normal distance from the front," which ordinarily is 15 to 20 or 30 miles.

"Two thousand sixty-one cases have been evacuated to North Africa by air and sea.

"The beach medical service was superior. One medical battalion distinguished itself on the beaches under heavy fire early in the operation. I shall recommend that that unit be cited for its gallant work under terrible conditions.

"The medical supply system began to function according to plan with the assault wave, and despite the most difficult conditions it rapidly developed to the highest state of efficiency.

"Among the difficulties with which the medical services have had to cope were the loss of the entire equipment of our third evacuation hospital and the bombing of a hospital ship," that was the British hospital ship which was lost in the Mediterranean, "which was bringing in nurses for the two evacuation hospitals that had been established. Fortunately, only one nurse was injured and all are again on their way to Italy to rejoin their units.

"The whole performance of the 5th Army Medical Service has been most heartening to me and has been of incalculable aid in the operation. I have been so favorably impressed with their performance that I cannot forbear to write you this personal letter to tell you of my gratitude and admiration.

"(Signed) Mark W. Clark, Lt. General,
United States Army,
Commander."

I had an opportunity to be in North Africa in late April and May and to visit our evacuation hospitals during the Tunisian campaign. They operated there as General Clark says they did in Italy, and General Eisenhower and General Clark both—I saw them both in North Africa—told me that the outstanding job done by any branch of the service in the Tunisian campaign was done by the Medical Department. (Applause) Those are your colleagues who are over there working.

The distances in North Africa are great. From Tunisia to Casablanca, our Atlantic base, is 1200 miles. This fighting was going on in hilly country with valleys between the hills, country that reminded one a bit of California, open valleys full of grain fields back to the rear where the fighting was going on, full of vineyards with great red poppies growing everywhere. But up front in Tunisia the ambulance drivers who were carrying back the sick and wounded were driving under blackout conditions over mountain roads fifteen, twenty or thirty miles. When they got out of the ambulances and unloaded, I was told by commanding officers of those evacuation hospitals, that they staggered like drunken men, from fatigue. They drank coffee and more coffee, got back in the ambulance and went up front for another load.

One division on the left flank in that offensive fought over hilly country, over mountain ridges with deep gulleys between. Wounded were carried by hand litter as far as fourteen miles. Litter bearers in evacuation hospitals carried stretchers from tent to tent until their hands were covered with blisters.

The nurses out there didn't have easy going either. They were sleeping in tents; they were doing their own washing by hand; they didn't have on pretty white uniforms that you are used to seeing nurses wear; they were wearing soldiers' coveralls, and to keep warm they were wearing soldiers' heavy underclothes, and to wade around in the mud from October until mid-May they wore soldiers' issue shoes. The mud was half ankle deep. They worked magnificently, without complaint. It was cold out there. They had anti-freeze in the radiators of the trucks last winter to keep them from freezing. When I was there in May I drove around in a jeep one afternoon, and I was cold when I ate dinner and I went to bed in a tent with all my clothes on and five blankets—I did take my shoes off—and I was cold all night.

The surgeons were doing a magnificent job. When the push was on they were working up to twenty hours in those evacuation hospitals. Auxiliary surgical units came forward offering their aid. The surgical teams would have none of them and said it was their job. That was the spirit.

For operating rooms they had ward tents. The floor of the tent was the wheat field the tent was pitched on that morning, and yet they were operating on heads and chests and abdomens. The mortality rate in those hospitals was around 2.5 per cent, which is a much better rate than we had in the last war.

To what can we attribute this low death rate? *First,* I must say, to plasma. The plasma has been made available to our armed forces through our Red Cross donor centers in this nation, and the blood of our people at home that has been processed and sent over as dried plasma. That has been available to the battalion aid, to dressing stations, to clearing stations; in fact, it has been available to give to the wounded man before shock develops. Prevention is always better than treatment. As much of it as is needed is given.

I met a doctor here today who is a member of this Association whose son was seriously wounded in Sicily. I think he had some six or eight vials of plasma that saved his life. Had that not been there he would not be back in America today.

Second, to excellent surgery. I think our surgeons that we have over there are technically superior to any men who have done work on a battlefield before; in fact, I know they are, and I am sure that our British colleagues' surgical work is equally good and they agree that ours is.

Third, to sulfa drugs, though sulfa drugs will never take the place of proper surgery, thoroughly debrided wounds, the removal of foreign bodies, the opening of deep fascia spaces, the leaving of those wounds open, and the proper splinting of compound fractures and large wounds before transportation, both before and after surgery.

Plasma was supplemented by whole blood transfusions, blood transfusion on the "hoof," so to speak, either from the detachment of the hospital that was serving the patients or from walking wounded. Every soldier carries his blood type on his dog tag around his neck. He is bled, the blood is cross-checked before transfusions are given, but blood is available in each evacuation hospital and general hospital to which these cases are transferred, either from walking wounded or from Medical Department enlisted men in the hospital.

I have another letter here from a British Major General who was on General Eisenhower's staff, who heads up that army there, and he writes me about his observations in the 5th Army. It was written the 5th of October.

"Dear General:

"I am taking a lucky opportunity. I am sending this to you by hand, happening to find myself on a special airplane flying back to Algiers from Naples and on to Washington. I have visited the 5th Army in the past few days. The forward divisional arrangements are magnificent. Officers and enlisted men are keen, skillful, courageous, earning Silver Stars freely. Transportation and equipment are splendid. In one division they have an exchange system of plasma, four bottles on each ambulance to replace twelve bottles held at the battalion aid posts. Also aid men are trained to put needles into veins. I saw a Silver Star sergeant" (that is once decorated, you know) "who goes up, does first aid, and starts plasma, which is continued on the way down to the battalion medical officer while the litter bearers are carrying the casualty. Another division always carries 900 bottles of plasma and keeps twenty to thirty with the battalion medical officer. I like this division plan.

"I hope a general hospital will soon be in Naples. The Corps in Army areas and evacuation hospitals are doing magnificent work. No American hospital refuses British or vice versa. The Corps Surgeon and the Army Surgeon are magnificent. Cooperation is perfect. You need have no anxiety of your Medical Department in the field; it is splendid. Heartiest relations exist between Command and Medical Department at all levels.

"Kindest regards.

"Sincerely,
"Cowell."

We might say a bit about head injuries. I think you have just heard a paper on that, but these head injuries are treated and given emergency care in our evacuation hospitals as soon as they arrive; chest wounds likewise. In a sucking wound at the front vaseline gauze is placed over it, or some tight dressing, strapped tight with adhesive and not sutured. If sutured, emphysema is liable to occur in the tissues and infection be found when the chest wall has to be opened for surgery at the evacuation hospital.

I was surprised to see how many wounds of the abdomen survived. The rule is that in perforation of the small intestine, the perforations will be sutured and resection will be resorted to only when necessary. The big bowel, if it is free, is to be brought outside. If it isn't free so that it can be exteriorized, it is closed by suture and a proximal colostomy performed.

In surgery of the extremities, I have enumerated thorough débridement and excision of the wound with proper splinting. Most cases are immobilized in plaster. For a time, skin type plaster was used. This caused interference with the circulation and gangrene. It won't do early in surgery with transportation to the rear. It was found that all cases must be properly padded and must be bivalved or slit immediately after the application of plaster, before transportation is begun.

There has been little or no gas gangrene. We have not been fighting in that type country yet. There is more in Sicily than there was in North Africa. Here, there were twelve cases with one death. That one death occurred in a case of gunshot wound of the buttocks which was improperly débrided, partially sutured and tightly packed, all of which was bad. The other cases were in those closed by primary suture or in cases in which circulation was destroyed, as in the muscles of the leg.

It has happened, as we knew it would, that no primary suture must be allowed in the front area in any compound wound and that packing is just as bad as suture.

We have had immersion feet on the West Coast from our action in Attu, with loss of toes and some feet. We found frostbite present in our aviators from high altitude flying when they had not been properly gloved.

In regard to evacuation, the Air Corps has done a splendid job. In Tunisia the patients had to be hauled 150 or more miles by ambulance to an airfield and some 15,000 were transported from there back to our first base sector, a three-hour evacuation by air. There were no railroads, and that evacuation would almost have been impossible with ambulances. These planes came forward bringing up supplies. The supplies were unloaded and arrangements made so that each plane would carry back sixteen litter cases and they were flown back within three hours to our general hospitals.

Air evacuation has even been done to a greater extent in the Southwest Pacific, and cases from Attu Island have been flown directly to the mainland from the hospitals in Attu. Some hundred thousand cases in all have been evacuated by air. For evacuation of wounded from North Africa home we have in operation now hospital ships that are protected by the Geneva Convention, and we must say that the Germans on the whole have respected the Red Cross. No strafing or bombing of hospitals has occurred in daylight when they have been well marked with the Red Cross. It takes a Red Cross on the ground a hundred feet square to be seen from the air at 20,000 feet. Why these British hospital ships were bombed we don't quite understand.

From the disease standpoint, you know our troops are pretty well protected from typhoid, typhus, small-pox, cholera, tetanus, and yellow fever by vaccination. We hope soon to have a toxoid for Welch bacillus to prevent gas gangrene. Our tetanus toxoid has been a hundred per cent effective; we have had no cases of tetanus. We have had three cases of typhus in North Africa with no death.

For insect-borne diseases we have no protection, except for yellow fever. We have none for malaria, for dengue, for diarrhea, for dysentery. These are all insect borne. The malaria mosquito in the Southwest Pacific has given us many, many more casualties as far as the non-effective rate is concerned than the Japanese bullet. The protection from malaria infection is a command function. Troops must be prevented from being bitten by the infecting mosquito. This is carried out by the use of proper clothing, not shorts but longs, to cover extremities. This mosquito bites at night. It is the female and she plays around nights in night clubs and such from sundown until sunrise. This is the time troops must be protected. There are certain repellents that can be rubbed on exposed parts that will keep the mosquito away for four hundred minutes. A bomb has been developed that contains Pyrethrum under pressure; a turn of a cockscrew and a tent or building can be sprayed, killing all the mosquitoes or flies in it. In the back areas, hospitals and quarters for troops must be screened to prevent malaria infection; likewise messes, to keep flies away from food, because feces, food and flies mean diarrhea and dysentery and loss from duty of manpower. Actually these are command functions and duties.

We have in the Southwest Pacific a disease with which we have not been familiar, known as scrub typhus. It is another type of typhus. It belongs to the same family but it differs from that which we see in North Africa and from that which we have at home. It is carried by a mite that lives on rodents and causes myocarditis and pneumonia. It is very toxic. There are high temperatures and a fairly high death rate. We have a commission now in the Southwest Pacific studying it. We also have a commission in Egypt studying typhus fever.

We have malaria in North Africa now. We have it in Sicily now, and we have it in Italy now. It is seasonal there, but we have it the year round in the Tropics. Up until now our worst enemy from days lost has been malaria, more than the Jap bullet or the Hun bullet.

I thought you might be interested in what we have at home to take care of these sick and wounded when they return, and the training that has been given these medical units getting them ready to go over there. There are now some 500 station hospitals and some 59 general hospitals in the United States with a total of over 300,000 beds. These general hospitals receive war casualties. They arrive at the ports at New York, San Francisco, Charleston, or elsewhere, and are taken initially to general hospitals at these ports, are sorted and distributed to other general hospitals. We have set up centers in these general hospitals for chest surgery, maxillofacial and plastic surgery, centers for the deaf; for the blind. There are five amputation centers and quite a few neurosurgical centers, as well as centers for vascular surgery.

I want to say a word about the dental service. It used to be a man had to have teeth to chew with before he could get in the Army, but there were so many men who didn't have them that we couldn't get an Army, so we took them without teeth. The only thing that keeps an enlisted man now from getting in the Army is an infection in his mouth of some type, an osteomyelitis, or something of that sort. We take them with no teeth at all. The dental cripple has been quite a job to our dental service, and it doesn't speak well for a nation when men under thirty-eight years old are edentulous. Twenty-five per cent of all inductees require emergency dental care and many of them have never seen a dental surgeon before. Sixty per cent require replacements and fillings. In August 1943 there were 2,000,000 fillings done in this Army at home, 430,000 extractions, and 76,000 dentures were made.

Now about these people who are doing this job and helping our doctors and nurses overseas, I might say that we have trained in this country 11,000 Medical Administrative Corps officers in our officer candidate schools. They are laymen who will replace that many doctors doing non-professional jobs in our hospitals in our forward units. In fact, there are some 13,000 commissioned in that Corps and in the Sanitary Corps. Sixty thousand enlisted technicians have been trained as medical, surgical, laboratory, x-ray, and dental technicians. Likewise, there have been thousands of clerks, cooks, auto mechanics and chauffeurs trained in Medical Department replacement training centers. We are running a school of tropical medicine at the Army Medical Center, as you may know, probably one of the best schools that has ever been operated in the United States. Colonel Strong of New York heads up that school.

There are other research problems going on. For instance, at Camp Knox they have chambers as hot as the Tropics, and as moist. I was in one last Sunday and I am sure it is. Men who have volunteered are in there taking atabrine to determine blood levels. They stay in there three weeks; they don't leave; they walk around and do certain exercise every day. Another group outside is working under similar conditions to determine atabrine levels of blood. Then there are cold rooms in which men sit, and they look as though they are dead, all huddled up with a lot of wires with needles attached which are stuck into their toes. One fellow's toes recorded a reading below zero.

The Air Corps has done an excellent job on high altitude flying, and research has been carried on to protect from combat fatigue and flying hazards. Initially, a tank was built and the soldier had to adjust himself, regardless of size, etc. Now, after medical research, the tank is being built around the man so that he may operate it effectively. Fire hazard and noxious gasses are likewise being studied and eliminated.

Rheumatic fever is quite a problem and a study is being made. There is a lot of it in certain parts of the West.

The Veterinary Corps is also a part of the Medical Department. Four hundred ninety million pounds of meat and dairy products are inspected monthly in the United States. Over 50,000 animals have been vaccinated each year against equine encephalitis. This vaccine was developed by that Corps and effectually prevents that disease.

There are some 11,000 war dogs in the Army that they are responsible for.

I hope you will excuse me for this brief and sketchy review of what the Army is doing for your son and your neighbor's son overseas, some of the training that was necessary for it, and what a grand job the doctors of the United States are doing as Army surgeons.

I thank you.

Treatment of Fractures of the Long Bones

By ROGER ANDERSON, M. D.
Chief of Orthopedic and Fracture Service, King County Hospital,
and Consulting Orthopedist, U. S. Marine Hospital, Seattle, Washington

The urgent need throughout the centuries in fracture therapy has been not for another new method but for a standardized method. Such a standardized technic is now made practical by a novel use of transfixions.

This standard method operates on three principles, reduction, immobilization, and maintenance of function. The value of the first two has been recognized for a long time. However, means of fully utilizing the advantages of these two principles was limited. The third principle, maintenance of function, never received much consideration as it was considered too idealistic.

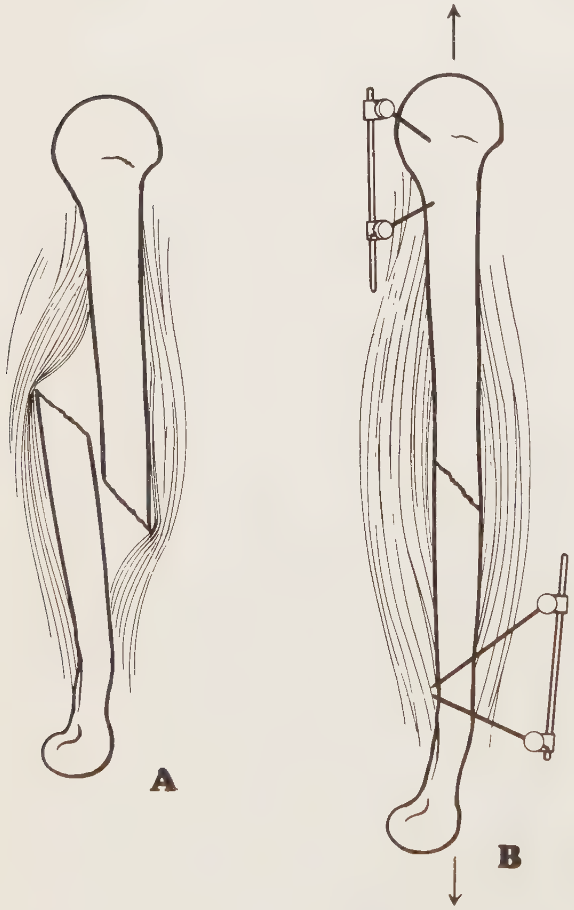

FIG. I. When traction is exerted in the longitudinal axis of the fragments with countertraction applied directly to the upper fragment, it will correct both the shortening and the general alignment. (For clarity only muscles on anterior and posterior sides of humerus are illustrated.)

A. *Reduction,* the first step in fracture therapy, is achieved by an entirely new concept of obtaining apposition. The problem of overcoming deformities was approached from a new basis. That is, instead of combating the muscles, the muscles were converted into friendly agents that would cooperate in achieving approximation. At first thought, such a premise may seem illogical, but the following figure illustrates how the muscles, when tensed in a certain manner, will actually mold the ends of the fragments into alignment. This method of conversion of muscles from antagonists to assisting agents is accomplished when the two requisites are employed: first, efficient traction in the line of the axis of the bone, and second, traction so applied that the fragments are free to move while under traction.

The above requisites (that is, efficient traction and free movement of the fragments thereunder) have not been obtainable in the past. However, when two transfixions are angularly inserted into each fragment, the most effective form of skeletal traction and skeletal countertraction is made available. Furthermore, dual transfixions supply such control that the traction force can be exerted directly down the anatomic axis of the fractured bone. Moreover, the fragments, even under traction, are free to move so that the muscles may mold them into alignment.

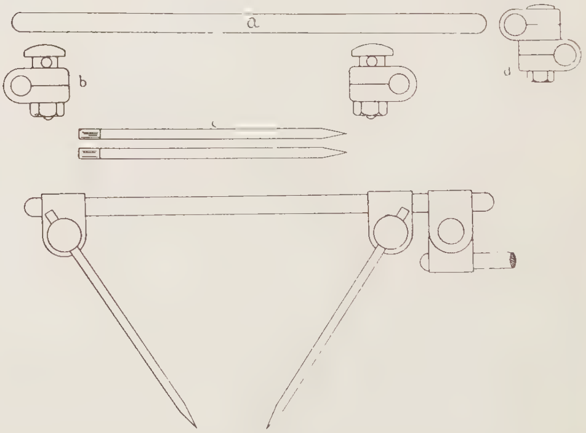

FIG. II. Two pins, angularly inserted into each fragment, furnish a triple function: First, a direct attachment for skeletal traction and skeletal countertraction; second, a handle for manipulative reduction; and third, a rigid anchoring device for castless or cast immobilization. Such fixation is so direct and positive that crutch ambulation with joint movements is feasible.

Achievement of end-to-end contact in itself does not mean reduction, as a full correction of rotational displacements must be accomplished before reduction can be said to be accomplished. Here again the muscles will assist in a correction of rotational displacement, provided the fragments are left free to rotate while under traction. This phenomenon is best illustrated by the following figure.

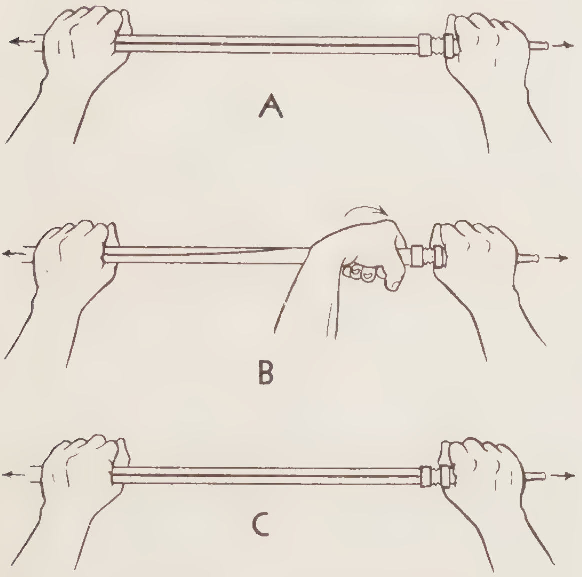

Fig. III. A. Correction of rotational displacement by an untwisting action can be demonstrated by partially screwing on the nozzle of a rubber garden hose and then exerting traction by grasping the nozzle with one hand and pulling in the axis of the hose, while the other hand pulls in the opposite direction for countertraction.

B. With the same amount of traction maintained, a second person twists the end of the hose close to the nozzle.

C. Note that while the same amount of traction is being exerted, when the twisting grasp is released the hose, being elastic, will twist back into the original position. This is made possible because the elastic hose is free to rotate at the nozzle.

Traction is employed not only to overcome the shortening but also to put the elastic muscles, the flexible fasciae, and the pliable skin under tension so as to mold the fragments into alignment. In general there are three ways of obtaining the traction force: (1) manual; (2) fracture table; and (3) self-aligning apparatus.

B. *Immobilization.* While the value of fixation in fracture therapy has been recognized for a long time, the means for a positive and complete immobilization has not been available by non-operative means. A plaster cast only partially immobilizes because as swelling recedes and atrophy occurs the cast is snug for a short period of time only. The only attempt ever made to directly immobilize the superior fragment has been by open operation and plating. However, with this dual transfixation method both fragments are controlled by a direct, constant, and positive fixation.

There are three ways of joining these two sets of transfixions for immobilization: First, by rod; second, by plaster which incorporates the transfixions; and third, by a combination of rod and plaster. This technic calls only for immobilization of the fragments as the contiguous joints are left free to move. Therefore, the patient may be fully dressed and crutch ambulatory with the assurance that each fragment will be positively held in position regardless of joint ambulation.

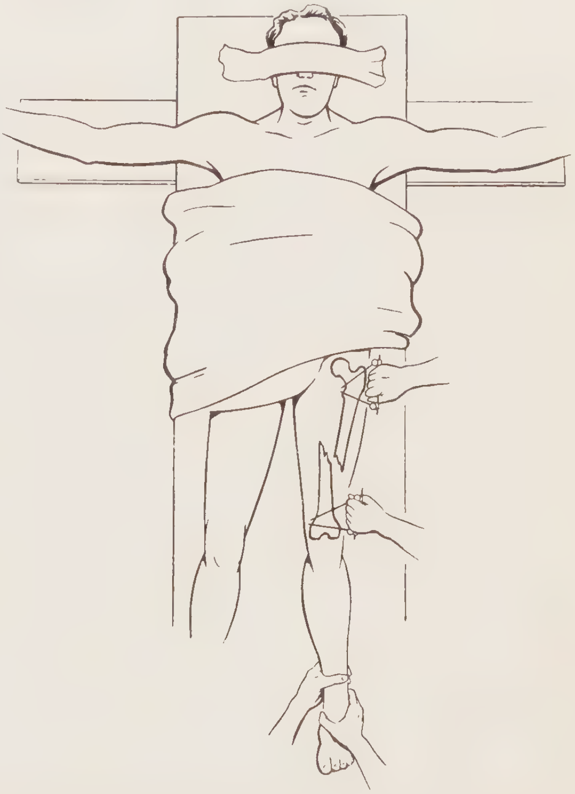

Fig. IV. Manual traction and countertraction, supplemented by manual manipulation of the fragments, is the simplest way of obtaining reduction.

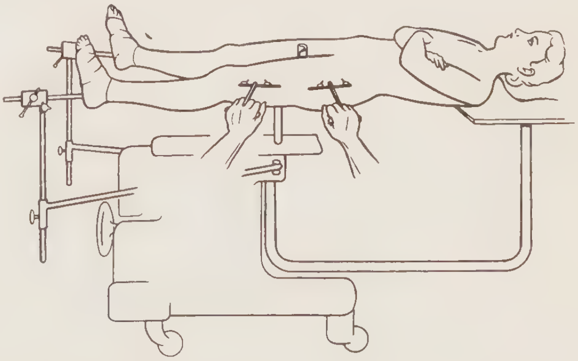

Fig. V. Reduction by these principles can be achieved on any standard fracture table, provided the distal fragment is rotated and manipulated back into its normal position, and that after traction in the long axis has been applied reduction is completed by manually manipulating the superior fragment into contact with the distal face.

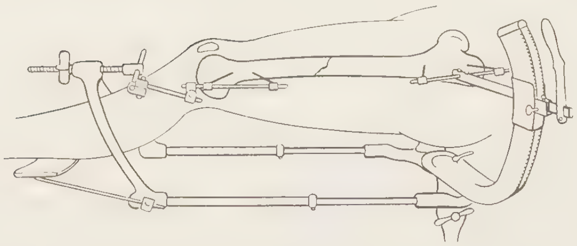

FIG. VI. Perfection of reduction can best be achieved by use of the free-rotating, self-aligning apparatus. Skeletal countertraction to the superior fragment is in line with the long axis of the fragment, leaving it free to move in both sidewise and vertical directions as traction is applied. The rotatory displacement of the superior fragment is first mechanically corrected and held in the normal or mid position of rotation.

The distal fragment is so held in the splint that even under traction this fragment can move easily in sidewise and vertical directions and also can be freely rotated upon the normal anatomic axis. This inherent ability of the fragments to freely move while the traction-countertraction force is being exerted permits the resilient tube of muscles, fascia, and soft tissues to mold the fragments into alignment.

C. *Maintenance of function.* Maintaining function in a fractured extremity may at first seem like wishful thinking but nevertheless with this transfixation technic this ideal is made practical. The economic advantages of such a method are readily evident, but the physiological advantages are as valuable.

It is clinically well recognized that when a limb is immobilized in a position of disuse, both the bone and the muscle atrophy. Another form of atrophy that has not been so well recognized is vascular atresia, which often expresses itself in later stages in the convalescence by a persistent swelling of the foot, ankle and leg. These atrophies are prone to occur in the recumbent patient with the leg immobilized in that position of disuse, elevation. Here most of the body functions have to go into reverse. Eating and swallowing and elimination are in the uphill position, while the injured extremity is at a much higher level from the supplying heart pump. Decreased activity, not only of the extremity but of the body as a whole, upsets both local and general metabolism. However, in a patient who is crutch ambulant, activities approach the normal. Muscle contraction calls for acceleration of the arterial stream while the contraction of the muscles upon the veins activates the venous return. This helps to maintain normal vascular activity and so, as the callus forming elements are derived from the blood stream, with more normal circulation the quicker and better the formation of callus will be.

Dangers and complications: While there can be little disagreement that this method possesses many idealistic advantages, the question may be raised as to safety and practicality. Some feel that it is a very radical and dangerous procedure and that it might result (1) in infection; (2) non-union; or (3) poor result. These fears are usually expressed by those who have had no personal experience with the technic. While these complications do occur when fractures are treated by this method or by any type of skeletal traction, fortunately they are preventable. Complications arise not from the use but from the misuse of transfixions. As the unguided scalpel can cause complications likewise can the mishandled transfixion cause complications.

1. *Infection,* the surgical spectre, is the result of a break in technic. The introduction of a transfixion, if properly performed, has no more septic potentialities than the insertion of a needle into a vein, a spinal puncture, or sternal infusion.

Ordinary pin seepage, erroneously attributed to infection, is a physical phenomenon attributable to one or more mechanical causes. The most common source of seepage is due to an irritation of the pin

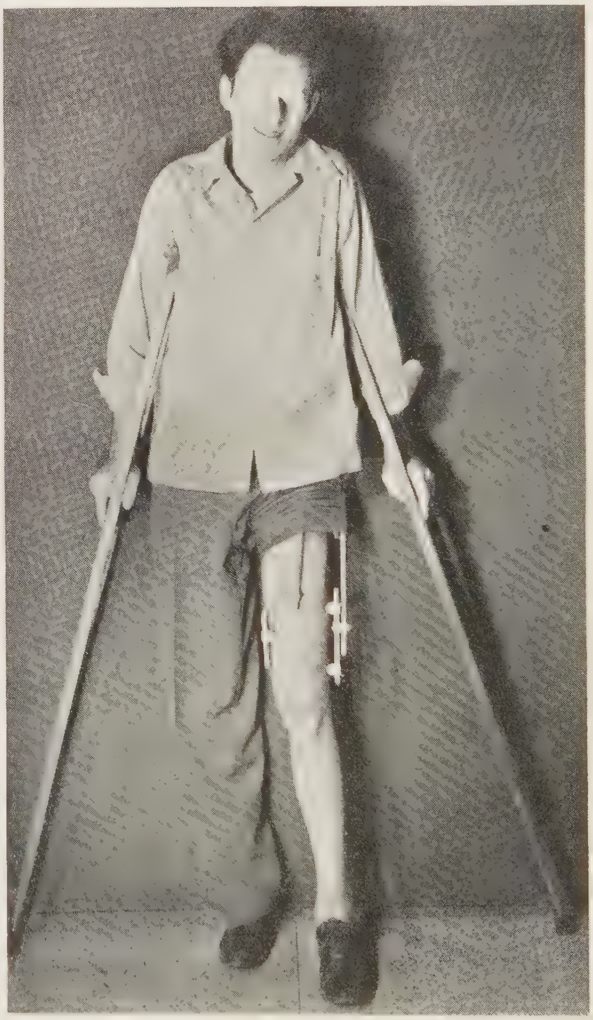

FIG. VII. Mr. W. K., compound comminuted fracture of the shaft that had been treated elsewhere so that it was not reduced until the second week.

wounds from a pulling of the skin against the transfixions. This type of seepage disappears on the removal of the pin. Another cause of seepage arises from fast drilling of the cortical bone with a dull or improper pointed pin. Such drilling causes sufficient heating of the point to cause a thermal necrosis with ring sequestrum formation.

Loose pins may be due to a hammering of the pins through the bone or excessive wobbling of the pin when drilling but are usually due to a failure to drill half-pins in far enough to penetrate the opposite cortex. Proper placement, drilling, and depth penetration of transfixions will remove most of the sources of seepage. Seepage usually comes from an irritation of the bone and of adjacent tissues from loose transfixions.

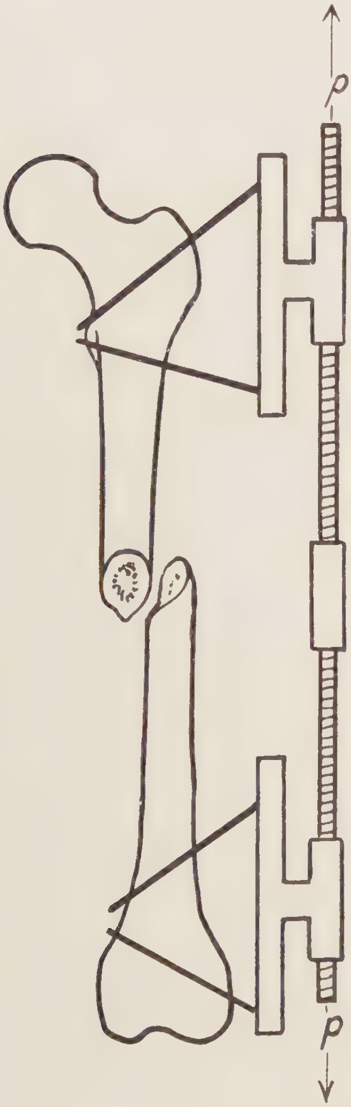

Fig. VIII. This type of splint is not capable of effecting reduction as it cannot rotate the fragments on their axis. In fact the design actually impedes such form of manipulation. Furthermore, the traction force is not exerted on the axis of the bone but is exerted on the external aspect so the fragments are not free to move while under traction.

2. *Non-unions*: Non-union is the chief danger in the use of transfixation fracture therapy. Even a slight space in transverse fractures will delay the union and in some cases may be the cause of non-union. However, if during the convalescence the transfixions are used not for traction but for contaction, unions will then be achieved in a quicker time than by any other method.

3. *Poor results*: Poor results, expressed in the form of incomplete reduction, loss of contact, angulation, painful immobilization, discharges, atrophies, and other complications can usually be attributed to the hustling enthusiast who, without preparation, rushes in to treat each fracture. If one is too busy to learn and to follow the transfixion commandments he would benefit the patient by referring him to a surgeon who knows and adheres to the rules.

Another prolific breeder of complications is one who attempts to substitute a splint for knowledge and ability, or one who blindly follows an untested technic advocated for the purposes of self-aggrandizement. Riding on the popular wave of acceptance of the advantages of this method of treating fractures, many opportunists, corporate and individual, are capitalizing on the general enthusiasm by marketing what they call a fracture reducing splint, when in reality the splint is not constructed to operate as such. The use of such equipment or modifications thereof, as illustrated in Fig. VIII, can only end in disaster.

Treatment of fractures by those who are not so inclined or who are without any mechanical aptitude has produced complications which have been erroneously attributed to the method. Successful clinical experience of hundreds of surgeons has demonstrated that complications arise not from use but from misuse of transfixions.

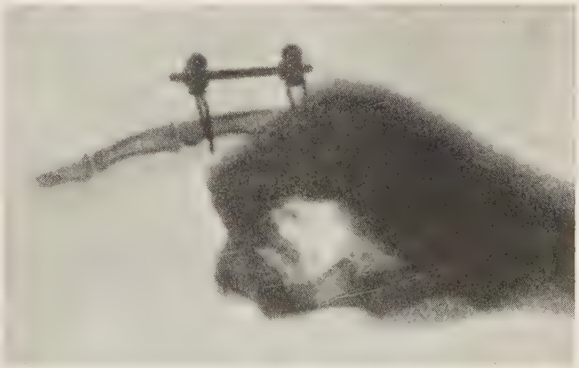

Fig. IX. Fracture of the smallest of the long bones, the metacarpal, can be managed with these principles as illustrated by a case treated by Dr. Lloyd Fisher, Oakland, Cal.

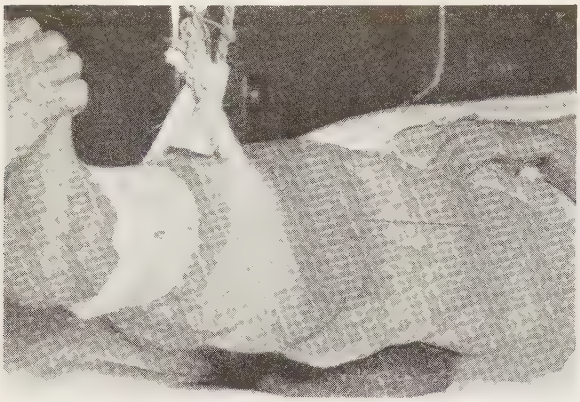

FIG. X. Mrs. P. C., age 57, weight nearly 400 pounds. Comminuted fractures of the tibia and fibula, even in obese individuals, can be managed as well with the standard technic as the fracture of the metacarpal shown before. (In spite of preexisting phlebitis, varicose veins, and a glandular imbalance, union was obtained in this fracture in about the usual time.)

RESUME

A standardized method of treating all shaft fractures, operating on the three principles of reduction, fixation, and maintenance of function, was described. Reduction problems are controlled by a different approach, as instead of devising means of combating muscle pulls, these same muscle forces were utilized to produce alignment. This self-aligning reduction technic is also a standardized procedure.

This new method is so inclusive that it can control any type of fracture of the long bones of either extremity, be they transverse, spiral, comminuted or compound. Furthermore, this technic is so comprehensive that it will handle equally well even fractures that extend into the knee, ankle, elbow or wrist joints.

The activation of these three principles, reduction, fixation, and maintenance of function, is made practical by the novel employment of transfixions in which two transfixions are angularly inserted into both the distal and superior fragments. These transfixions supply a positive means for skeletal traction and skeletal countertraction, besides furnishing handles for the direct manipulation of each fragment for reduction and supplying a positive means for immobilizing both fragments. This standard method of treating all types of fractures of the long bones, besides providing for crutch ambulation with contiguous joint function, supplies several essential physiological advantages and many evident economic advantages.

Rocky Mountain Spotted Fever; An increasing hazard; Diagnosis and Treatment

By GEORGE E. BAKER, M. D.
Casper, Wyoming

Studies on insect borne infections have gained additional impetus in the past few years. Typhus fever and Rocky Mountain spotted fever are members of this group. The former has occasioned concern because of the present worldwide conflict, again placing mankind in danger of plaguelike dissemination. The latter, while less distinctive in its capabilities is nevertheless a disease of serious potentialities. It may in time become nationwide in distribution. For that reason it should command attention from all of us.

Rocky Mountain spotted fever is often called "spotted fever" or "tick fever" for the sake of brevity. Although the number of cases remains insignificant, resultant mortality is cause for considerable speculation. Of the 450 to 600 cases diagnosed each year in the United States, nearly one-fourth terminate fatally.

Tick fever was long considered to be a curiosity of American medicine. Supposed localization stimulated little interest on the disease in sections of the country other than western endemic areas. Medical schools paid it scant heed in their teaching curriculums. They often included discussions of it with those of tropical diseases, although it has nothing in common with them.

Identification of the first cases of the disease in the East led to a storm of controversy and disbelief. It was generally assumed that reported cases were in reality typhus fever because of similar clinical manifestations shown by the two infections. In 1931 the disease was proved to be present in other sections of the country, far removed from its supposed habitat. Physicians slowly became reconciled to its general distribution. It was soon realized that we possessed little factual knowledge on tick fever, textbooks of Medicine paying it but cursory attention. It became necessary for western investigators to clarify the picture. It is due to their efforts that much accurate information on tick fever and tick borne infections in general has been placed at the disposal of the medical profession.

Practicing medicine in the western endemic area, in a section of the country where tick fever occurs with considerable frequency, I have had occasion to make study of its various aspects. The disease in this particular locality is moderately severe to severe in intensity. Seldom is a case of mild intensity seen. As fatalities which result from the infection closely parallel in number the severity of existent illnesses, deaths must be expected. For a period of time from 1927, during which accurate statistics have been kept by the Wyoming State Health Department, 1101 cases have occurred in the state, with 218 deaths. For the same period of time 151 have appeared in our (Natrona) county, with 30 deaths.

For the reason that nearly every fifth individual stricken with tick fever is doomed to succumb to the illness, members of the local medical profession, residents in the community and surrounding rural areas are intensely concerned over the disease. The subject of Rocky Mountain spotted fever is extensive. I shall make no attempt to deal with its numerous phases, but instead shall present those of major importance for your thought and consideration.

The disease is not one primarily of human beings. It is one of animals. Ticks are not parasites of the human race. When individuals are bitten, the occurrence is an accident. The wood tick, Dermacentor andersoni, is vector in the western endemic areas. The common dog tick, Dermacentor variabilis, is carrier in the eastern and midwestern sections. Recently the lone-star tick, Amblyomma americanum, has been identified as a third vector in certain of the southwestern states. Transmission of tick fever by other species is apparently possible, because with making allowance for different life cycles and host habits of other types, maintenance of the disease is explained satisfactorily wherever it exists. Many others have been incriminated, serving either as carriers or both as carriers and transmitters of it to human beings. The potentialities of the majority of them are not perfectly understood. In every region of the country in which the occurrence of tick fever is a possibility, native species have been isolated and their characteristics in part at least identified.

The cause of tick fever is generally agreed to be a micro-organism. Dermacentroxenus rickettsii is gram

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Acknowledgement is made to Drs. R. R. Parker, N. H. Topping and H. R. Cox and others for authoritative information, and to Dr. W. Francis Smith for the kodachrome moving picture film "Rocky Mountain Spotted Fever," used to illustrate the article.

negative. It is best identified in infected tissues by means of modified Giemsa's stains. Rickettsias are small, intracellular, coccoid or bacillary bodies observed in infected tissues. They can be cultivated only on living tissues, attempted growth on mediums free from living cells being unsuccessful. They represent a relatively new type of micro-organism, bacterium-like in character, displaying the same characteristics of staining shown by spirochetes.

Although tick fever occurs more frequently in men because they are most often exposed to infection, neither sex is immune. The infection strikes individuals of all ages. Apparently all races are susceptible to its ravages. The greatest number of cases occur in groups of the population engaged in outdoor occupations, principally agriculture. Persons living in range areas, such as those handling sheep are in danger. Hunters and trappers, prospectors and miners, forest service workers, survey crews, highway construction workers, railroad section hands and those who only occasionally visit infected areas for reasons of business or pleasure such as fishermen, picnickers, campers and tourists are also affected. The disease is prevalent among unprotected laboratory workers, as a result of handling engorged ticks which can easily rupture and so spread their highly infectious contents.

The disease appears to have a cyclic tendency. More cases are reported some years than others. The reason for this trend is unknown. It is believed to depend upon local and regional conditions. The number of individuals exposed, the abundance of ticks, the percentage carrying infection and the capability of the virus to produce frank infections from year to year seems to play a part. The possible relationship between prevalence of ticks and animal hosts is another factor.

In the western section of the country the tick season begins early in the spring, usually around the middle of March. It continues for approximately three and a half months and tapers off around the first of July. In the mountainous regions the highest incidence of tick fever is during the late spring months, owing to delay in the advent of warm weather. In eastern areas the disease is more prevalent in the late spring and early summer months, but cases can occur in the fall of the year. In these regions the tick season extends from April until late August or early September.

The appearance and disappearance of ticks is governed by conditions of moisture and temperature. They may appear earlier one season than another and likewise disappear earlier. Wet damp weather makes them less active. As a result they are less apt to seek hosts. Warm dry weather brings them into full activity, so that they attach themselves readily. When ticks emerge in the spring from hibernation, their virus virulence is low. If subjected to incubation or allowed to ingest blood, they gain the ability to bring about frank infections. The phenomenon is called "reactivation." It accounts for the observation that but few cases of tick fever are seen for a variable period of time following appearance of ticks in the spring. After they have lain dormant all winter the infectiousness of virus carried by them is at a low ebb. With advent of warm weather, the vectors become activated and quickly infectious. As a result, the number of cases of the disease multiply as the season advances. In any locality tick fever can occur in colder months of the year, granted that there is abundant warm sunshine or adequate conditions of artificial heat. These factors release ticks from their dormant state, reactivating them so that they move about in search of hosts.

The virulence of tick fever varies greatly in different localities but appears to remain fairly constant for any one region. Reasons for the variance are unknown. It is supposed that repeated passage of the virus through successive animal hosts play a part. It is justifiable to speak of mild, moderately severe or severe types of the disease, in view of the great difference in virulence of the infection in various localities and sections of the country.

The percentage of ticks that carry infectious virus has been studied mainly in the wood tick. It varies in different localities and from year to year in the same locality. Frequently it is less than 1 per cent. It rarely exceeds 5 per cent, although in small, localized areas it has been reported as high as 11 per cent. However, even in areas known to be heavily infected the ratio of infected to non-infected ticks is admitted to be very low.

Tick fever has a usual incubation period of from 4 to 8 days. The prodromal manifestations are not dissimilar to those from other febrile states, there being malaise, headache, loss of appetite and chilling sensations. These vary in degree and last two or three days.

The onset is usually abrupt, initial symptoms often appearing in the late afternoon or early evening, when the day's work is completed. A definite chill is common, associated with persistent frontal headache and severe aches and pains in the muscles, bones and joints. The latter are more pronounced in the back and lower extremities. Pressure over the calf muscles or free movement of them invariably elicits pain. Inspection of the stricken individual does not usually reveal the presence of crawling or attached ticks, it being customary for him to remove them before reporting for medical care. Indurated sites of former attachment may be detected. They are not unusual in appearance, but are occasionally discolored from subcutaneous blood extravasation. The regional lymph nodes may be palpable and tender.

An initial elevated, macular rose colored eruption is sometimes present. Its presence is not distinctive. It is not until the typical maculopapular eruption appears that the first clue regarding the nature of the illness is given. It is seen 24 to 48 hours after the onset of tick fever. Often spoken of as being petechial or "flea-bitten" in appearance, it is customarily first detected on the wrists and ankles. The eruption can easily be overlooked in its early stages, particularly in individuals of dark skinned races or workers much tanned on the extremities from overexposure to the elements. Within a period of a few hours it becomes sharply demarcated and spreads from the initial locations in a centripetal fashion over the chest and abdomen and thence to the remainder of the body. The eruption is always more marked on the extremities than elsewhere. Extension is complete in two or three days. The associated generalized aches and pains are then partially relieved, but temperature remains high.

The maculo-papular eruption is considered to be the most characteristic finding in early tick fever, but diagnosis of the disease must not depend on its presence. Mildly affected individuals or those previously vaccinated never show a rash or an insignificant one. Severely afflicted patients often die before its appearance from generalized toxemia. Occasional victims of the disease have atypical or bizarre eruptions. The lesions do not disappear on pressure except during the early stages. They are accentuated by tourniquet application. Lesions may eventually involve the palms of the hands, soles of the feet and the mucosa of the inner cheeks and throat. The completely erupted patient is truly speckled or spotted, having a rash which covers the entire body. Petechiae may appear in successive stages of the disease. Each crop-like flareup, of which there may be three or four, lasts approximately fourteen days.

The eruption tends to remain discrete in milder cases of tick fever. It is first rose red and later bluish red. Discreteness does not persist in more severe ones. The petechiae increase in size. Confluency and coalescence follow. A mass of purpuric areas may involve the entire body. If terminal gangrene ensues, sloughing of the soft palate, scrotum or other dependent body parts gives the afflicted individual a pathetic appearance.

The petechial eruption gradually fades as patients recover. The process takes much longer in severe cases than in mild ones. It occurs with the fall of temperature. Desquamation may occur, bran-like in character or so complete as to result in casts of body parts. Pigmentation remains at former petechial sites. It may be followed by the formation of minute cicatrices. For months after recovery from tick fever temporary manifestations of the eruption are brought out by overexposure to heat or cold. These often cause the patient intense concern. They last only a short while, clearing when normal skin temperatures are re-established.

There is an abrupt rise in temperature within the first 24 hours of the onset of the disease. But one or two slight remissions occur, a fastigium of 103 to 105 F. being reached by the beginning of the second week in mild cases, by the second or third day in more severe ones. With recovery from acute manifestations of the illness, temperature falls by rapid or slow lysis, rarely by crisis unless the case is an abortive one. Slight temperature remissions may occur in mild cases, but fever is constant to slightly rising in more severe ones. It is sometimes distinctly remittent after the first few days, particularly in moderately severe protracted cases, but never ceases until terminal lysis has occurred. The temperature may be normal from the first or subnormal in very severe forms of the disease, to rise sharply in the 24 hours preceding death, or it may be high from the first, then drop to normal and rise sharply again before death occurs. An uneventful drop of temperature to normal, followed by a secondary rise without apparent justification, should suspicion the development of complications.

The pulse is of good volume early in the disease. It is slow, averaging 90 beats a minute. Early disproportion of the pulse and temperature ratios is one of the characteristic findings of tick fever at its onset. Loss of strength and volume of the pulse occurs when myocardial weakening ensues from toxemia. It rises out of proportion to the temperature. Cardiac involvement results in blood pressure fall, the first heart sound becoming muffled and indistinct.

The respirations at first are normal or but slightly increased. In severe cases they accelerate, the change accompanying alterations of the pulse and temperature ratio. Increased rates often signify the development of pneumonia.

For the reason that these are considered to be the most typical findings in tick fever, I have dwelt at considerable length on them. There are other manifestations. They exist in various combinations, their intensity often depending on the severity of the disease process.

Patients moderately or severely ill with the disease show marked prostration. All senses are dulled. Mental confusion is present, even though afflicted individuals appear rational to superficial examination. Amnesia is often troublesome, persisting until the eruption is complete or for some time afterward. One is impressed by the anxiety and concern displayed by sufferers. The cheeks are flushed and the eyes injected. Photophobia is sometimes extreme. There may be nervous disturbances, such as lethargy, restlessness or nervous irritability. Children are subject to convulsions. Insomnia is at times troublesome. Active delirium can occur, particularly in severe cases during terminal stages of the illness. Muscular twitchings and fibrillatory tremors are common. Muscle tonus is definitely increased throughout the disease. Aches and pains may persist over the body. Distress occasioned by them may be agonizing. When located in the muscles of the abdomen, an acute surgical condition can be simulated. Slight stiffness is often detected on movement of the neck.

The tongue is swollen and moist early in the disease. It becomes dry and coated in severe cases, having a darkened border and prominent papillae. When profound swelling ensues it often protrudes from the mouth. If coma occurs it becomes fissured and covered by sordes. A dry hacking, non-exudative cough accompanies pharyngeal engorgement. It indicates bronchial irritation. Profound chilliness is often present. It is not shaking or chattering in character, but tends to be most persistent and drawn out, frequently lasting for a period of from two to four hours.

The skin is sometimes tender. Patients may complain bitterly of pressure from light bed coverings or drafts of air. It becomes dark red or bluish in severe cases, color changes being most evident on the back and thighs. When patients are examined under satisfactory light conditions an ill-defined bluish discoloration is commonly seen beneath the skin surfaces. Dependent body parts may slough. The process usually involves the scrotum or soft palate. Necrosis can occur, affecting the prepuce, toes, fingers or ear lobes. Permanent alopecia sometimes follows the disease.

Anorexia is present. Nausea and vomiting take place in some cases, the regurgitated material at times containing blood. Diarrhea occasionally occurs; the stools may be bloody. Constipation is usual, often being most obstinate and difficult to overcome. Severe cases show loss of sphincter control. The spleen is invariably enlarged and tender, the liver sometimes demonstrating similar findings. There is jaundice. It tends to definitely increase in terminal fatal stages of the illness.

There may be an inability to void as a result of increased muscle tonus. At times there is incontinence. Urination can be quite painful. A lessened secretion of urine sometimes occurs, caused either by changes in the kidney or a failing circulation. It is frequently accompanied by edema. Total repression of urine formation can occur at the end in fatal cases of tick fever.

The total red blood cell count and hemoglobin content are lowered later in the disease. The white blood cell count is usually 12,000 to 15,000. It may reach 30,000. A relative mononucleosis is common, the average being 10 to 12 per cent.

The urine shows increased specific gravity. It may be highly colored. Albumin is passed in varying amounts by old or debilitated persons, acetone and microscopic alterations frequently being present as well. Urinary changes are not so often manifested by younger persons or those who have previously enjoyed good health.

Spinal fluid findings are not significant. Blood chemistry studies in tick fever have never been conclusively worked out. Examination of blood smears for the causative micro-organism, while sometimes successful in detecting it, does so with such infrequency as to make the procedure worthless.

The three most common complications of the disease are bronchial or lobar pneumonia and phlebitis. Hiccup, hemorrhages from the nose, intestine or kidneys may occur. There can be intercurrent conditions, due directly or indirectly to ticks. Streptococcic septicemia, tularemia and tick paralysis may co-exist. Development of sequelae from the infection is possible. Deafness, impaired vision, insomnia, neurosis, anemia and tachycardia occur. Partial intestinal obstruction has been reported. The disease often leads to rapid progress of degenerative processes in aged individuals. Some persons who have recovered from very severe infections seem to lack their former endurance. In occasional instances they show lessened mental acuity.

Tick fever can be confused with various other infections, particularly when the disease appears unexpectedly in a locality or if encountered by those unfamiliar with its manifestations. The diseases most commonly causing error are typhoid fever, measles, scarlet fever, smallpox, postmeasles encephalitis, purpura hemorrhagica, epidemic cerebrospinal meningitis, secondary syphilis, Colorado mountain fever and endemic typhus fever. Epidemic typhus fever while as yet a sporadic disease in this country, at some future date may enter into the differential diagnosis. In recent years, since the sulfonamides have become popular, their use in varied febrile states has resulted in eruptions which often closely simulate those found in tick fever. Confusing diseases can usually be ruled out by careful histories, examinations and repeated observations of infected persons, together with confirmatory laboratory studies.

The diagnostic procedures ordinarily employed are the infection test, the protection or virus neutralization test and the Weil-Felix agglutination reaction. The latter is perhaps the most popular and widely used at the present time. A new test which may be of even greater value is the complement fixation test, using the specific antigen of tick fever. The test, if carefully done, will differentiate between typhus fever and tick fever in most instances. It becomes positive about the same time as the Weil-Felix test, but sustains its high titer for a longer period of time, perhaps for a period of years. More complete references to the procedures may be found in other papers.

Prognosis in tick fever depends on the ability of infected persons to withstand ravages of the disease, particularly in reference to myocardial and renal intoxication. Bad omens are those of a confluent purpuric eruption, with terminal sloughing, definite temperature and pulse reactions, severe intoxication of the brain and central nervous system, and the development of complications, particularly in older or debilitated individuals or those ill with intercurrent conditions.

Treatment is purely symptomatic and supportive in character. An attitude of helplessness and hopeless inactivity on the part of those caring for tick fever patients cannot be tolerated. Carefully directed symptomatic care and supportive measures assist patients in every means possible. Vigorous yet well directed procedures bring about successful outcomes in many whose recovery seems hopeless at the time they are first placed under care.

With the onset of the illness bed rest and good nursing care are imperative. Strength must be conserved as much as possible. Patients should be kept quiet, both mentally and physically. Baths, packs and simple sedation often accomplish this. If codein or morphine is indicated, they must be used as freely as necessary. Cold or tepid bathing is dangerous. It results in shock to severely ill patients. Bath temperatures should be 70 F. or above to be safely tolerated.

The gastrointestinal tract needs careful watching. The diet should be adequate and nourishing, yet easily digestible. Fluids must be given freely. Oral administration is preferred. When there is excessive vomiting they can be supplied parenterally. Regular elimination is assured by mild enemas or cathartics. Frequent urinary examinations are necessary. They detect pathological alterations at their onset.

Should myocardial weakening appear imminent, support to the heart is indicated. Skin care is important. Equal parts of witch hazel and alcohol in water applied once or twice a day as a sponge often comforts and invigorates severely ill patients. It removes soreness from muscles and revives them. They are less mentally dulled, appearing stronger for several hours following the procedure. Mouth hygiene is essential. Oral antiseptic mouth washes, varied in order to give patients a change, rid the region of accumulated waste products. As a result, sufferers are made more comfortable during acute phases of the illness.

The use of convalescent serums and transfusions are without beneficial effect. Some physicians resort to autohemotherapy, 10 to 20 cc. of citrated blood from the patient being readministered intramuscularly. The procedure is repeated as often as necessary. Drugs of the sulfonamide series have little or no value in the management of tick fever. Evidence would seem to indicate that their action is an unfavorable one. If pneumonia, phlebitis or other complications of a like nature occur, their use is justified, the drugs of choice depending on the invading microorganism. Tick vaccine must never be used for treatment. It has no beneficial action when used for this purpose. In milder cases its use is too drastic to be justified; in more severe ones it may prove dangerous as regards ultimate recovery.

Recently Topping has produced a specific antiserum. It has been shown to be effective in treating laboratory animals infected with tick fever. Cases of the disease have received this material and, although the results are not conclusive, mainly because of the relatively small series, they are sufficiently encouraging to warrant its use. Similar to other specific antisera, it must be administered as early as possible in the course of the disease, preferably before the fifth day of the illness. There is no evidence that the serum is of value if given later than the fifth or sixth day of fever. Until the present it has been administered intramuscularly in divided doses, the total being approximately 1 cc. per kilogram of body weight. The anti-Rocky Mountain spotted fever serum is now commercially available.

Symptomatic and supportive measures offer much in treatment, but unfortunately they are not sufficient in themselves to insure recovery. In 1934 the late Dr. J. C. Kamp of Casper and I began original investigations on the use of neoarsphenamine dissolved in aqueous metaphen solution administered intravenously. We had previously received encouraging reports of results obtained by use of the two drugs in combination for treatment of endemic typhus fever. That year we used them on nine moderately severe cases of tick fever, without deaths from the illness. Response was so gratifying from administration of the chemotherapeutic agents that we decided to use them in the future as a routine measure in all cases which came under our supervision.

Since that time an average of three to four cases of the disease have been under my care each season. During the past nine years all cases so treated have recovered. Local physicians and ones practicing in other sections are using the two drugs in combination as an adjunct to care for individuals seen by them. Although it is impossible to evaluate their responses, those whom I have communicated with report them to be equally gratifying. Many of them join me in the firm conviction that neoarsphenamine in metaphen solution is a definite therapeutic aid in the treatment of tick fever.

I do not think that they exert a specific action on manifestations of the disease. Their approach is admittedly uncertain. Benefits derived from their use may be credited to direct action on rickettsias in tissues. They can be attributable to a combination of the bactericidal action of metaphen together with the spirocheticidal action of neoarsphenamine on a microorganism which is bacterium-like in character, yet has staining properties at least similar to those displayed by spirochetes. It is certain that those so treated show less evidence of intoxication, minimal damage to the heart and kidneys and a more discrete, brighter colored eruption. The rash does not become hemorrhagic and is usually more sparse in distribution. There are milder nervous symptoms and less mental depression. In fact, the clinical picture is less alarming in every way. Convalescence is more rapidly established, being of shorter duration with a minimum of complications. None of the patients had received tick vaccine for the purpose of immunization against the disease.

Although the number of cases treated with neoarsphenamine in metaphen solution is not impressive, if satisfactory responses continue to be received in the future, one can anticipate far reaching consequences. Epidemic typhus fever is a dreaded disease, but a rickettsial infection strikingly similar in many of its clinical manifestations to tick fever. Inasmuch as use of the two drugs in combination have proven successful in treatment of endemic typhus fever and tick fever, is it too much to expect that the combination might exert beneficial action on epidemic typhus fever as well? Only time and thorough trial can give the answer.

In performance of the procedure .3 gram of neoarsphenamine is dissolved thoroughly in 10 cc. of an aqueous solution of metaphen, dilution 1:1000. The resultant yellow and turbid mixture changes but little on standing. It is warmed and injected slowly by vein, the same precautions being observed as with any chemotherapeutic agent intended for intravenous administration. Solution is administered and blood alternately withdrawn into the syringe until the entire amount has been given. The procedure usually consumes a period of five to ten minutes. No reactions, local or constitutional, either immediate or delayed, have thus far been observed.

Administration of the two drugs is repeated at three to four day intervals. Three or four injections have customarily been sufficient to ameliorate the clinical picture so as to insure ultimate recovery. Continued or recurrent manifestations would apparently justify additional administration of neoarsphenamine in metaphen solution.

Should a case of the disease demonstrate severe renal injury as a result of the infection, careful consideration must them be given to the question as to whether or not their use is justified. The inherent risks associated with drugs of considerable potency on an already damaged kidney must be weighed against benefits to be derived from their administration. I secure first morning specimens of urine for careful examination on the day the material is to be given. These have never shown sufficient alterations to suggest severe renal disease. I feel that renal damage which accompanies tick fever is avoided by prompt and regular administration of neoarsphenamine in metaphen solution. It may be that is why I have not encountered evidences of severe kidney pathology. It has never been necessary for me to omit administration of the drugs at the time scheduled for their use.

Tick vaccine protects against tick fever. The material is prepared by the Rocky Mountain Laboratory of the National Institute of Health, Division of Infectious Diseases, at Hamilton, Montana. It is dispensed to physicians desiring it for the purpose of immunizing those who run the danger of being exposed to the disease. Tick vaccine is made in two types. The older one is from tick tissues and the more recent one from embryonic chick tissues. Chick embryo type has not superseded the vaccine prepared from ticks, and, although it is less likely to cause reactions, evidence regarding its immunizing value is not so certain.

Recommended dosage of tick tissue vaccine for those who have never previously been vaccinated is 2 cc., repeated at an interval of from seven to ten days. If the particular locality is one in which serious cases of tick fever are known to originate, the second injection must be followed by a third, administered after the same time interval. Children receive a proportionate amount of material, 1 cc. being recommended for those ten years of age or younger. Dosage of the chick embryo type is slightly different, in that three injections of 1 cc. each, administered after the same time interval, are recommended. For individuals who have been vaccinated each of the past three years, two injections of 1 cc. each of either the chick embryo type or chick tissue type are suggested.

Intramuscular administration of vaccine is not known to bring about more than a slight constitutional reaction. The same precautions must be observed as with the injection of any biologic product intended for an immunization procedure. There is an immediate sense of fulness at the site, which is followed by one of smarting or stinging. Itching may occur, exacerbated by scratching or rubbing the part. Generalized malaise is sometimes noticed, often with a slight febrile reaction. The manifestations are usually transitory, subsiding before subsequent administrations of the material. These usually result in much milder symptoms or none at all. Reactions to the use of vaccine have not been so prevalent in recent years. It has been purified and standardized, so that the optimum dosage of the material is better known.

The degree of protection afforded by vaccine and its duration varies with vaccinated persons and the virulence of infection to which they are exposed. Those vaccinated in the spring of the year usually retain a considerable degree of immunity for at least the remainder of that year. They are usually afforded full protection against relatively mild strains of the disease, but are progressively less protected as virulence of the virus is increased. Nevertheless, even against the more severe forms of tick fever protection is usually adequate to ameliorate materially the usual stormy course of the infection so as to insure ultimate recovery.

It is probable that a certain proportion of individuals carry an indefinite degree of immunity into the second year, even against highly virulent strains of virus. The degree of protection appears to be greater in those who have been vaccinated for two or more successive years. Evidence does not indicate that any considerable degree is carried into the third year. It is recommended that immunization be performed each year in order to afford the greatest degree of protection possible.

Exposure to infection is avoided only by remaining out of tick infested localities. Business or pleasure activities may make the precaution impossible. Those entering regions where ticks abound should wear trousers, gathered by some means at the bottoms in order to prevent vectors from crawling up the legs. Contrary to popular belief ticks do not jump on those who pass their vantage points, but lie in wait on short grass or vegetation not over 18 inches above the surface of the ground, actively moving their serrated legs, by which means they seek transfer to objects that brush by. Smooth clothes prevent ticks from gaining footholds; those of rough texture impede their progress. All clothing should have a minimum of seams and openings in order to prevent their ingress to body surfaces. For the reason that ticks often gain access to the body by working themselves beneath the collar it is a good plan to occasionally pass the hand around the neck to detect their presence.

Clothing should be removed at least two or three times a day and a diligent search conducted for crawling or attached ticks. The procedure must be a thorough one. They usually hide away in body folds, crevices and hairy portions free from rubbing. Camps should be located where rodents are few, preferably in places where no low grass, sagebrush or small bushes are growing. Wooded areas along creek banks are best avoided, as are the vicinities of old trails and roads. The safest camping spots are usually where standing timber is present with a minimum of low vegetation. Before retiring for the night in the open, campers must again inspect their persons, clothing and bedding. The precaution is of added significance when two persons sleep in close proximity. Infected ticks may attach themselves to both successively. The first one may escape infection or be but mildly ill, the second one more seriously so from reactivation of virus in the tick vector by blood ingestion from the first victim. While in tick infested localities it is unwise to leave bedding spread on the ground during the day. It attracts ticks, often from a considerable distance. After return from trips, clothes and bedding should be immediately and carefully inspected, aired and then removed to buildings not used for human habitation. Eradication of ticks which have taken up their abode in a location is a most difficult and uncertain procedure.

Ticks move slowly about for a variable length of time after getting on the body, during which they seek suitable locations for attachment. The process is not noticeable to victims. They are usually not aware of crawling ticks. It is well to seek out the cause of minor irritations, because these may indicate their presence. Little reliance can be placed in the contention that vectors of the disease are not actively infectious until several hours after attachment has occurred. When located, attached ticks must be removed without delay. Removal is a procedure requiring considerable skill and perseverance if it is to be safely accomplished. As a rule the head of the tick is embedded beneath the surface of the skin, the body remaining free and protruding at an angle from it. The head is held firmly in place by mouth parts. Hasty or careless plucking often serves to remove the body alone, leaving the remainder in place to serve as a potential source of infection. Gentle traction may be successful in removing the tick. Close inspection then reveals it to be intact, often with a small fragment of epidermis caught in the mouth parts.

If it is found that the head is too deeply embedded to be removed by gentle traction, the point of a scalpel or hypodermic needle can be inserted under it, and removal effected by turning the point upward. A small piece of epidermis in which the tick's head lies embedded may be elevated with a pair of tweezers and a tent-like wedge of tissue cut off with a razor blade or snipped with a fine pair of scissors. Either maneuver is accomplished in a matter of seconds and insures safe and complete removal. Resultant wounds from tick extraction must be thoroughly cauterized, using phenol, silver nitrate, iodine or similar agents. A light sterile dressing should then be applied.

Removal of engorged ticks with bare hands is a dangerous practice and should not be indulged in. Care must be exercised so as not to crush them. If the accident occurs, the discharged contents should be thoroughly washed from the hands by soap and water, care being exercised not to irritate the skin. Virus is apt to be highly infectious, even on unabraded skin surfaces. Precautions for its removal are most important, it being through neglect of them that many otherwise inexplainable infections have resulted.

Summary and Conclusions

Epidemic typhus fever and Rocky Mountain spotted fever (spotted fever, tick fever) are disease entities of serious potential significance. They are rickettsial infections, similar to one another in clinical manifestations. The former, as a result of the present conflict, has possibilities for world-wide dissemination. The latter, because of as yet undeterminable factors, has commenced to show rapid distribution over the United States. Neoarsphenamine in aqueous metaphen solution administered intravenously is meeting with considerable success in treatment of an increasing number of cases of tick fever. It is hoped that the two drugs in combination can be given a thorough trial in epidemic typhus fever, in order to determine whether or not they are equally effective for treatment of that dreaded disease.

In conclusion, I would like to show the following excellent kodachrome moving picture film entitled "Rocky Mountain Spotted Fever," prepared by Dr. W. Francis Smith of Lander, Wyoming. It should bring out by illustration some of the points which I have stressed in previous remarks.

DIAGNOSTIC SURGICAL CLINIC

The Management of Breast Tumors

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I thought it might be wise to present the subject of the management of breast tumors, when I was asked to choose a topic. It has been my experience that there is a good deal of confusion in the minds of many men and women as to just what attitude to take upon breast tumors. I think these conflicting emotions, if you want to call them such, of indecision and the lack of some definite procedure often lead the doctor into a state of bewilderment, and I think very often the patient suffers thereby.

In the beginning I should like to lay down just a few dicta or premises upon which we base our conclusions and our procedures. The first is that we believe that every solitary (I mean solitary in the sense of single, not multiple) breast tumor should be surgically removed without delay. There may be some question in the child and even in that case we are beginning to feel that it is a wise thing to do. Every solitary tumor should be removed without delay.

Secondly, that so far as cancer of the breast is concerned we believe the one procedure is a radical mastectomy, not temporizing with any other type of procedure, providing, of course, we believe the tumor or the case is curable. The procedure should be radical enough to completely eliminate the disease, and I stress this especially because I think when we try to bring in the aid of irradiation we may feel that if we do not do the operation any too thoroughly the irradiation will take care of it. I believe that is a fatal attitude to take. The operation must be radical enough that we completely sterilize the field so far as carcinoma is concerned.

There has been considerable talk relative to local mastectomy in the treatment of carcinoma of the breast. I do not believe that we are alone in our attitude of severely condemning it. The correct procedure is a radical mastectomy. That of course brings up the treatment by irradiation. We have had considerable experience with this sphere of activity, that is the possible sterilization of the field by the use of irradiation. I might say that in every instance in which we have treated the patient with x-ray for the reasons that I shall give you, following irradiation—and when I say "irradiation" I mean giving from 4500 to 6000 r, enough to bring about a very pronounced irradiation dermatitis or reaction—even though we could palpate no tumor or axillary nodes in every instance we have found carcinoma still existing in the specimen after the operation has been performed.

If you don't find it, it is not because it is not there; it is because you do not look carefully enough. We have a case now which illustrates this. A woman had a rapidly growing tumor and was given massive x-ray treatment. The tumor entirely disappeared, all the axillary nodes disappeared; the operation was delayed several weeks because of furunculosis that involved the chest wall. When she was operated upon the specimen was examined three different times by the pathologist who reported to me that there was no carcinoma found. When I took issue with him and told him if he was just a little more persistent he would find it, he came forth two days later with two positive findings, one in a gland and one in breast tissue in which there was carcinoma. That demonstrates why we take such a pronounced attitude.

Thirdly, we should divide our carcinoma cases into those that are curable and those which should have palliative therapy. That applies, of course, to carcinoma anywhere in the body. Do we believe the case is curable and shall we use curative procedures, or is the case inoperable and incurable and shall we institute palliative procedures? This is important.

Fourth, we use x-ray only in the exceptional case, either preoperatively or postoperatively. In the so-called inflammatory type of carcinoma, rapidly spreading in the young individual usually shortly after labor, I think surgery dooms the individual. They die very promptly from recurrences or spreading of the tumor. In that type we use irradiation, and in a few others in which the disease seems to involve considerable skin, one of those spreading, rapidly growing types, we use it. We use it only in those cases. We do not believe that preoperative or postoperative irradiation in the average case increases the percentage of cures, reduces the time of remissions, or the number of remissions.

With these premises in mind, these cases are managed in the following manner: When the patient comes in we evaluate the condition. I will not have time to go into the various diagnostic points. We take into consideration, of course, inflammatory diseases. Once in a while an abscess may be confusing. Tuberculosis naturally will be because it is a chronic type of disease which has a tendency to form a rather hard, fixed tumor mass. Duct obstruction with low-grade infection occasionally may be confounding. Several times we have found ducts dilated to a half to three-quarters of a centimeter filled with pasty to solid material, or sometimes with a turbid liquid. Fibrocystic disease or what is commonly termed chronic cystic mastitis, a term we do not use any more, will at times enter into the picture. Then, of course, benign and malignant neoplasms and unusual conditions such as Paget's disease and fat necrosis; Paget's disease is included in carcinoma of the breast.

We try in every instance to arrive at a tentative diagnosis and we put forth every effort that we may say this is so-and-so. In the clinic, if there are any differences of opinion we ask each man to formulate his own and it is written on the history sheet and he signs it. I think when a man signs his name to a statement or to an opinion he is much more likely to study his reasons for giving the opinion. Therefore, we try to make a diagnosis preoperatively in every case. We do not use the aspiration biopsy. I do not condemn it, but I believe it is only of importance when it is definitely positive. A negative biopsy means nothing. I wonder sometimes whether the average biopsy is always reliable.

There are correlated findings which help us sometimes to arrive at a diagnosis. I have brought such a patient here because she presents a tumor of the breast that has some of the qualities and characteristics of carcinoma if you look at it only superficially. It is possible we cannot demonstrate this to you, but she has in the median inferior quadrant of the left breast a tumor mass that is firm, and if you were close enough you could see that it dimples the skin. We pay a great deal of attention to fixation and dimpling of the skin. I believe if there is one characteristic symptom of cancer of the breast it is dimpling of the skin. In one instance I made a diagnosis of carcinoma without feeling any mass whatsoever, solely because there was a dimple in the skin. This was excised and the carcinoma, probably not over 3 or 4 millimeters in diameter, was found in the underlying tissue.

This lady has in the median inferior quadrant of the breast a tumor mass which dimples the skin; it is irregular and firm, and when she presented herself at the clinic we wondered whether it might be a malignant process. The interesting thing is that when you look further, beyond the breast, she has a very similar process at the elbow. We all know what this it. It is a simple lipoma. She has others. The fact that she has a lipoma of the arm and several others makes it quite obvious that this woman has a lipoma of the breast. She can have a lipoma of the arm and a carcinoma of the breast, but here is a tumor in the breast exactly like the others. This will be excised, and I am certain it will be found to be a lipoma.

After the patient has been evaluated and generally studied from the standpoint of a complete physical examination and ordinary routine laboratory work, the situation is then explained to the patient: "You have a lump in your breast which is a tumor and it should be removed." We tell the patient that the tumor will be locally removed and studied. If it is a benign tumor the breast is closed. If it is a malignant tumor a radical mastectomy will be done at once. This is all explained to the patient. The patient is then brought into the hospital with a tentative diagnosis of definitely benign tumor, as far as we know, a definitely malignant tumor, as far as we know, or a questionable tumor.

The benign and questionable cases are prepared exactly as though a radical mastectomy would be done; in other words, the axilla and chest are shaved. We use only soap and water in preparation, followed by sterile water rinse; the whole chest wall and arm shoulder and neck are prepared as though we expected to do a radical mastectomy. Then an incision is made into the breast and the tumor widely excised. The tumor is then inspected in the operating room by the surgeon and the pathologist. Let me say that in practically all our breast tumors we have our pathologist see the breast before the tumor is removed. I do not believe that we as clinical men take advantage of the pathologist as much as we should, and often we handicap him. We bring a specimen to him in the laboratory and expect him to make the diagnosis. He should know about the case beforehand.

In the largest percentage of cases the diagnosis is made without frozen section, I would say in nine or eight out of ten. Nevertheless, in all cases a frozen section is examined. If the lesion in benign the breast is repaired, and let me stress this point: we can remove a fairly large tumor from a woman's breast, repair the defect in the breast and leave practically no deformity. We often tell the students that we remove a benign tumor of the breast which requires probably five or ten minutes and then spend a half hour doing a repair of the breast.

I should like to illustrate what I mean. Here is a young lady who came to us when she was only nineteen years old. I want you to look at her breasts and see if you can see any evidence of an incision. (The Speaker asks which breast it is.) She has no obvious scar. This girl had a tumor mass under the areola on the left side. It was a good-sized affair, at least 3 or 4 cm. in diameter. It was lobulated; you could see it by rolling the skin back and forth. The incision was made following the junction of the areola and the skin, about half of the circumference; we reflected the areola and nipple upward, enucleated the tumor and repaired the defect with fine white silk, laid the areola back down and sutured it in place and it left her without any deformity or scar that is perceptible. Don't feel chagrined, Mr. Speaker, because I showed this patient to a group of medical students on Monday and they all asked the same question; standing right in front of her they wanted to know which breast was involved.

Six months after this enucleation she returned with a recurrence, apparently one little lobule was overlooked. We again opened the breast, extending the incision a little further medially, again elevated the areola and the nipple, enucleated the tumor, repaired the defect, and again replaced the areola and nipple, and here she is without any deformity, with a perfectly normal breast.

I think that this has an agreeable effect upon the individual, and I think, gentlemen, that if we will do these things women will be less reluctant to have tumors removed than if you leave a markedly disfigured and unsightly breast.

In the average case, when we make a radial incision, after the tumor is out and find that it is a benign lesion, we start at the base of the wound, I mean on the pectoral side, using fine white silk and a small, curved cutting needle—we have to use a cutting needle because the breast is tough—and start, layer by layer, putting in white silk tier by tier bringing the breast tissue together until we reach the cutaneous border. Depending upon the size, we use sometimes five or six sutures; we have used as high as fifteen or twenty or twenty-five. We reconstruct the breast to prevent any spaces where blood or serum can accumulate. We intend to obtain complete apposition of the breast tissue. It is a perfectly amazing thing how the breast can be reconstructed after removing what appears to be a quarter of it, so that as you look at the breast there is no deformity as compared with the other, though possibly it may be a little smaller. It leaves a very sightly organ rather than one with a depressed ugly scar. If the tumor is proven to be malignant a radical mastectomy is performed.

So much for the benign tumors.

With unmistakable carcinoma of the breast the case is studied from the standpoint of the stage of the disease and its spread as may be demonstrated by x-rays of the chest, spine, skull and the pelvis. The findings will guide us as to the curability and operability of the individual case.

The presence of axillary nodes does not contraindicate a curative procedure. I think it was Harrington who stated that in a large series of primary operable breast cancer cases studied, in 29 per cent of the series that had no detectable axillary nodes, carcinomatous nodes were found when the specimens were examined. On the other hand, in 32 per cent in which axillary nodes were detected preoperatively they were found not to be carcinomatous. Therefore, the presence simply of axillary nodes must not deter us in making every effort to cure the patient. Naturally, those cases that have bone or visceral metastases are not curable and should have only palliative treatment.

We follow a definite plan of procedure in doing a radical mastectomy. We have done this for years and all the men that I have trained use it. We believe that it offers the best opportunity of eradicating the disease with the least danger of locally implanting cancerous cells.

After preparing the skin the patient is draped. The tumor forms the center of the skin and underlying tissue to be excised. The lines of incision are marked by a scratch of the back of the tip of the scalpel. The incision should extend downward from a point just median and below the acromion process, to encircle the breast and ends just below the costal margin. We attempt to remove that portion of the skin and subcutaneous tissue that lies between the tumor and the anterior axillary border on the theory that the lymphatics may contain tumor cells.

After the skin incision is marked, the breast is covered with a towel. The upper end of the incision is deepened and skin flaps undercut. The fat is dissected off the upper end of the pectoralis major muscle, the dissection being from above downward. The insertion of the muscle is identified, its lateral margin freed and a finger is passed under the insertion of the muscle. Clamps are placed and the muscle cut across, avoiding the clavicular portion. The muscle is then split in its longitudinal axis downward and medianward and drawn downward by means of a Lahey forceps placed on the cut end. The pectoralis minor is then isolated lying median and behind the cut end of the major muscle. Its margin is freed, a finger slipped behind it, clamps placed and the insertion of the muscle cut across. All the muscle should be included so as to be able to complete the axillary dissection later. Lahey clamps are placed on this cut muscle and it is also drawn downward. This procedure should uncover the entire anterior portion of the axilla.

The axillary fascia is then opened high up so as to uncover the branches of the brachial plexus. All areolar tissue, fat, small vessels and nerves are dissected away carrying the dissected mass intact downward. All small vessels are freed of fat about their junction with large vessels, clamped with small forceps, cut and then ligated, fine silk being used. This dissection is carried downward, posteriorly, medianly and laterally so as to completely free the axilla from the point where the vessels exit from the thoracic cage to well lateral in the mesial side of the arm. The only nerves besides the brachial plexus that are preserved are the long thoracic and thoracodorsal. I wish to emphasize one important point, i. e., constant complete hemostasis. We ligate every tiny vessel so as to have constantly a dry field that allows one to recognize arteries, veins and nerves without difficulty. The axillary fat pad should be freed from its attachments without entering the pad except at the margins. As the dissection progresses the axillary contents are drawn downward by forceps.

When the axillary dissection is completed the area is irrigated with warm saline solution and a gauze pack moistened with warm saline solution is placed in the axilla. The towel is then removed from the breast, the line of incision is completed, skin flaps are under cut and the breast with the underlying pectoral muscles is removed. All bleeding points are ligated with fine silk and the skin flaps are drawn into place. A stab wound is made in the posterior flap and a Penrose drain is inserted to extend up to the apex of the axilla. The skin is then closed with silk.

We are meticulous about the dressing. The arm is in full abduction. Fluffed gauze is placed over the wound and surrounding area and over the gauze are placed from four to six sea sponges moistened with warm saline solution. These sponges are so placed to press the skin flaps against the chest wall, care being taken that the skin is pressed well up in the axilla and counter compression anteriorly. The sponges are covered by pads and bound snuggly against the chest wall by stockinet roller bandages. The entire dressing is then fixed by wide strips of elastoplast. This dressing encases the patient in a very snug yet elastic dressing. Most patients complain of the tightness of the dressing but the discomfort is not severe. We have a feeling that if the patient does not complain, the dressing is not correct.

The primary dressing is not removed until the 5th postoperative day. At that time the drain is removed, a few of the stitches are cut and another compression dressing is put on. This latter dressing is removed on the 8th postoperative day, all stitches are removed and if the wound is healing satisfactorily an ordinary dressing is put on. Occasionally a pocket of bloody serum is found. This we attribute to an incorrect dressing that did not supply adequate compression. The fluid is aspirated and another compression dressing is applied.

Very few modifications of this procedure are employed. Occasionally the line of skin incision must be changed or the closure altered to meet specific conditions. The points to be stressed in the operation are: (1) constant complete hemostasis, (2) complete cleansing of the axilla without entering the axillary fat pad, (3) an adequate compression dressing. The amount of skin removed depends upon the extent of skin involved. On numerous occasions we have examined microscopically the "pig-skin" type but have never found carcinoma cells. In practically every case a primary closure is possible and skin grafting is resorted to only in rare cases.

In order to obtain free use of the arm, the patient is instructed to use the arm immediately after the operation. It is insisted upon that she place her hand behind her head. This is not difficult since the arm is dressed in full abduction. All actions involving the shoulder joint are insisted upon and within a few days the patient can carry these out without pain or discomfort.

There are few contraindications to the performance of a radical mastectomy. It must be conceded that in the presence of carcinoma of the breast the patient has a disease that will destroy her in a relatively short time. If the patient has some other disease that will surely destroy her in less time then the operation is not indicated. It may need to be postponed because of some acute infectious disease such as an upper respiratory infection; however, this means only a delay in the procedure. In the presence of far advanced cardio-vascular-renal disease the operation may not be justified; however, I have performed a radical mastectomy in a women who had a systolic blood pressure of 300, had had a cerebral hemorrhage previously and was believed to have a thoracic aneurism. She tolerated the operation remarkably well and made an uneventful recovery from the operation. Age is not a contraindication in itself. We have repeatedly performed the operation in patients in the seventh and eighth decades without untoward results. We have performed the operation in the presence of diabetes, hypertension, heart and kidney disease without accident.

So far as our mortality is concerned, I cannot give you any definite percentage. I do not know how many cases we have operated upon during the past twelve years but during this time we have no death following a radical mastectomy, therefore the mortality must be very low. It must be conceded that much depends upon variable factors inherent in such an operation. Chief of these are the length of time required to perform the operation, the preparation of the patient and the amount of blood lost during the operation. Our average time is one hour and twenty minutes, the operation having been done in fifty-two minutes. We are meticulous in hemostasis, therefore, our patients lose but little blood.

There are few complications. There should be no residual edema of the arm. Occasionally we note a slight edema for a month or two but this disappears. The edema that develops late and that is progressive is caused by recurrence or progress of the disease within the thorax about the large vessels and is a bad omen. Every patient should have free and complete use of the arm. Only too frequently we see a marginal necrosis of the wound. This is characterized by necrosis of the margins of the skin and often takes a very bizarre form being at times rather sharply demarcated and extending quite a distance into the skin flap. The cause so far as I know is not known. We thought for a while it was caused by the skin flaps being too thick, the fat preventing prompt fixation of the skin to the chest wall and preventing the formation of vascularization of the skin. Following this theory we cut the flaps very thin removing practically all the subcutaneous fat. Soon it was discovered that this was not the answer to the problem since marginal necrosis again occurred. It nevertheless seems logical that the flaps should be thin, trauma avoided and adequate pressure dressings applied.

As a summary I should like to stress several points in the management of breast tumors.

1. Whenever examining a woman for any purpose examine the breasts carefully and make a record of the findings.

2. Be suspicious of every breast tumor.

3. All solitary breast tumors should be promptly removed and studied grossly and microscopically at the time of operation.

4. A radical mastectomy is the only procedure for the cure of cancer of the breast. Never depend upon the assistance of irradiation to justify an incomplete or inadequate operation.

5. The breast should be completely repaired after the local removal of a benign tumor.

6. Neither preoperative or postoperative irradiation in the average case will improve your final results. They are an economic and physical strain upon the patient that is not warranted. Moreover the skin will have become sensitized so that if irradiation may be needed later for recurrences it cannot be used.

7. Following breast operations a compression dressing with sea sponges will facilitate prompt healing of the wound and prevent the accumulation of blood and serum.

8. Early free movements of the arm are essential after a radical mastectomy.

9. There are few contraindications to a radical mastectomy.

10. If the radical mastectomy is done correctly the mortality rate is very low. It should be one per cent or less.

DIAGNOSTIC NEUROLOGICAL CLINIC

Progressive Muscular Dystrophy

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Any discussion of the progressive muscular dystrophies tends to leave us with a sense of frustration because of the unfavorable therapeutic outlook in these types of cases. However, accurate clinical diagnosis will enable us to use our therapeutic methods to the best advantage. I wish to expand the title slightly to include examples that are not always included in this category. Three patients will be presented. One represents a typical case, that is, typical in most respects, of progressive muscular dystrophy. Then another has progressive muscular dystrophy and an associated condition which has been reported but rarely in the literature, the so-called family periodic paralysis. Finally I have a third who represents a different type of dystrophy from that usually included in the group, namely the dystrophy of Charcot-Marie-Tooth.

This lady you see here is now twenty-eight years of age. She came to us at the University of Illinois some two or three years ago with the story that at the age of twenty-three she had begun to notice difficulties in walking. Her legs seemed to be weak and, as she expressed it, stiff and achy at times when she walked or used her legs. This condition progressed gradually and became worse until, during the past two or three years, she has begun to fall repeatedly. This is the sum total of her complaints, but upon neurological examination it was found that she also had weakness in her upper extremities. I will demonstrate some of this for you.

The first thing you notice is that she cannot arise from a chair unaided; one has to help her up. Once she is on her feet, though, she does fairly well.

Watch her when she sits down. She has to hold herself to keep from falling into the chair. She has marked weakness also of the arms and the shoulder joints, but her grip is excellent and the power in her ankles is also quite good.

She has a biceps reflex. The knee-jerk is barely obtainable. This reflex you cannot see because it is so slight, but her ankle-jerk is, if anything, brisk. There are no pathological reflexes, and the remainder of the neurological examination is essentially negative.

When we go over this woman carefully we notice that her weakness is much more severe in the proximal portions of her four extremities. On the other hand, she has practically normal power in the distal portions of her extremities. This is a characteristic feature of the typical progressive muscular dystrophy.

One also notices that her calves are well developed but when she is stripped for examination there is some atrophy, especially around the shoulders and around the hips and in the glutei. The reflexes, likewise, as we have seen, are diminished proximally and not diminished, are practically normal, distally. There are no sensory changes and no pathological reflexes.

The symptom of pseudohypertrophy is not well illustrated in this patient and that is just as well because it frequently is absent in this disease.

Our next patient can walk perfectly well. This man has come in from quite a distance to be here, as have all the patients. He is now twenty-seven years of age. He came to us first five or six years ago, at which time he complained that in 1929 he began to have attacks of weakness of his whole body from his neck down. These attacks of weakness came on usually early in the morning, about three or four or five o'clock; they were apt to come after he had been exposed to cold and wet or after he had eaten a large meal the evening before. These attacks paralyzed his entire body from his neck down. He could move his eyes; he could swallow; he could talk and see and hear and observe everything, but he was completely paralyzed in all four extremities. His attacks usually passed away in from two to six hours, after which time he would be perfectly normal again. These attacks came on sometimes three or four or five times a week and nothing was found in his experience that would prevent them or ameliorate their severity when they did occur.

In addition, he noticed some slight weakness, even between attacks, although he thought so little of it that he did not mention it until he was asked about it. This weakness was found in his shoulders and in his hips. It is difficult to demonstrate this weakness before such a large group because it is so slight.

If he pushes his extended arms downward, although he is a well built, muscular man, he exhibits considerable weakness in his upper extremities proximally; in the lower extremities it is not so obvious.

He has rather a well developed physique despite this weakness and there seems to be some tendency toward pseudohypertrophy.

His tendon reflexes are sluggish over his whole body. There are no sensory changes, no pathological reflexes, no other neurologic abnormalities.

We admitted him to the Research and Educational Hospital of the University of Illinois and studied him on several occasions. Every possible measure, including prostigmine, glycine, ephedrine, various types of vitamin therapy, and so on, was tried without any benefit. He was observed in several of his acute attacks of paralysis. It should be mentioned that in these attacks he presented a most unusual neurologic picture in that not only were all his reflexes absent but his muscles had lost their entire electrical stimulability. They were not stimulable either by faradic or by galvanic current. He presented, therefore, the so-called cadaveric electric reaction, just as if we had tried to stimulate a cadaver. So far as I know, this absolute electrical refractoriness of the muscles is present only in this condition.

As the attack cleared up spontaneously, the electrical stimulability of the muscles gradually returned, but galvanic and faradic excitability returned pari passu.

We classified this condition, with its typical history of recurrent attacks of transient paralysis and the unique findings that we obtained during the attacks, as so-called periodic family paralysis. There was no one else in his family who had had this condition, but his trouble was so unmistakable that there could be no doubt about it.

Some three or four years ago it was discovered that a very simple procedure would abort or prevent these attacks. That was the administration of potassium in some form. So we gave him potassium chloride, which is very easy to give since it tastes almost the same as sodium chloride. It was given to him in aqueous solution. He was instructed to take one or two teaspoonfuls of the powdered crystals three times a day. When he did so, the attacks ceased entirely. How many years has that been?

PATIENT: About three years.

DR. MACKAY: For about three years now he has never had an attack unless he has exhausted his supply of potassium chloride or stopped it for experimental purposes, as we have had him do on more than one occasion.

In addition to this family periodic paralysis, he has progressive muscular dystrophy, very slight in degree, not worth having him disrobe here before you to show, because there would be so little to see. That association of family periodic paralysis with progressive muscular dystrophy has apparently been reported only once or twice in the literature.

Our third patient you will notice has a high steppage gait. He has to lift his feet high in walking because he has a foot-drop on both sides.

Would you mind taking off your coat and shirt down to your waist?

This man is now twenty-one years of age and single. His mother has the same trouble. He first told me that his difficulty was present when he was born, but that is scarcely true because as a matter of fact when he was a boy, and before he was twelve years of age, he ran with other boys and apparently was normal, although he was never good at athletics. When about twelve years of age he began to have real symptoms, consisting of weakness in the distal portions of his extremities. His hands became weak, his ankles and legs became weak, and the condition progressed steadily as the years went on. At the present time he has a complete foot-drop on both sides. He has marked weakness of his calf muscles on both sides, but the proximal segments of his extremities retain their power to a practically normal degree.

You see that he is a well developed young man. He has a very good muscular development, but no pseudohypertrophy. However, you can see, I am sure, that he has the typical atrophy of the small muscles of the hand with the *main en griffe* that one also sees in progressive muscular atrophy. His calf muscles are likewise quite atrophied, as you can see quite easily. His tibialis anticus groups are likewise atrophied.

There are no sensory changes in this patient. I might ask him if he has ever had any pain in connection with this trouble. Have you ever had any kind of pain, such as neuritic or rheumatic pains in your legs?

PATIENT: Not that I know of.

DR. MACKAY: Very often patients with this type of disturbance, i.e., a familial atrophy coming on early in life and distributed peripherally in the extremities, have sensory features associated with their motor weakness. These sensory features are chiefly vague shooting pains and gradual loss of sensibility. He has a marked reduction of vibratory sensibility in his ankles, but very little superficial sensory loss.

In examining this man's extremities we find no palpable enlargements of his peripheral nerves. In some of these cases, or closely similar cases, we find hypertrophy of the peripheral nerves, and on microscopic section one sees a so-called hypertrophic interstitial neuritis. There is debate as to whether hypertrophic interstitial neuritis and this patient's condition are really identical, and yet I think they are essentially the same. This man has a disease which has been given the name of Charcot-Marie-Tooth progressive muscular dystrophy. It, like the progressive muscular dystrophy of the usual type, is familial, but it differs in that the weakness is peripherally distributed rather than proximally; there is never any pseudohypertrophy; the reflexes disappear, as in the other type; and sensory changes are frequently associated with it.

I think we might allow our patients to go while we conclude our remarks.

I shall not consume your time in a discussion of pathology. I am sure you are familiar with the major features. I will say only that in progressive muscular dystrophy of the pseudohypertrophic or usual type there is no pathologic condition except in the muscles; in fact, this disease is thought to be due to a disturbance in the muscle itself, hence a primary myopathy. The nature of it, the cause of it, is totally unknown.

In the Charcot-Marie-Tooth type, the third patient that I showed you, the disease is different in that the sensory changes, the peripheral distribution of the weakness, and the pathologic findings of hyperplasia of the fibrous tissue in the peripheral nerves demonstrate that it is not a disease of the muscle itself, hence not a primary myopathy, but due rather to disease of the peripheral nerves. Hence it has been called a neuropathy or a neuritic type of progressive muscular dystrophy. That is about all we know about it; we do not know the cause of it; we do know that it is of familial incidence. It, therefore, has to do with the inherited characteristics of the individual. It must be represented in the genes of the germ plasm or represented by an absence of some of the genes in the germ plasm. Further than that we know nothing in detail about it.

We know even less about the cause of the attacks of family periodic paralysis which were exemplified in the second patient whom I demonstrated to you. A word about the chemistry of that condition is instructive. We find that these patients exhibit a consistently low level of potassium in the blood—5 or 6 milligrams, instead of the usual 10 or 11. There is moreover a great variability in the level of blood potassium. In general, they tend to have an especially low blood potassium during the attacks, and they never have attacks if the blood potassium is kept up anywhere near normal. However, they may have attacks at certain times when the blood potassium is higher than at some other times when they do not have attacks. In other words, there is no absolute correlation between the degree of reduction of blood potassium and the occurrence of these attacks.

A final word or two as to therapy. I am sorry to say that we have no treatment that is worth the proverbial tinker's dam in the treatment of progressive muscular dystrophy, either of the type that I first demonstrated, with proximal weakness, or the neuritic or peripheral type. We have the duty, nevertheless, to treat these patients; we must treat them for the purpose of keeping up their morale and enabling them to carry on as best they can under their difficulties. Consequently we do give them various substances. Of late, it is the fashion, as in every other branch of medicine, to give them vitamins. A few years ago, vitamin E and in particular that fraction of vitamin E called alpha tocopherol was introduced for the treatment of another type of muscular atrophy, namely, amyotrophic lateral sclerosis. Unfortunately, the original optimistic reports regarding the use of vitamin E have not been borne out by our experience with this disease, so that we now doubt whether it has any good effect—at least I do. After it was introduced in the treatment of amyotrophic lateral sclerosis, alpha tocopherol was then used in the treatment of these other conditions, again, so far as my observation goes, without any benefit whatever. So if any of you are under the impression that vitamin E or alpha tocopherol is of any value in these conditions I hope your experience bears you out, but I am afraid you are going to be disappointed.

Fortunately in those rare cases of family periodic paralysis (and, incidentally, it is not so very rare; in the past four or five years perhaps three or four of these cases have come to my own attention) we have something that is apparently absolutely specific, namely, the use of potassium. The easiest way to give potassium, as I have stated, is to give potassium chloride, as we did to this boy. If you wish to do so, you can have him place the potassium chloride crystals instead of sodium chloride in a salt shaker on his dining table. If his food is then prepared without salt, he can use the potassium chloride as a seasoning. It is a very satisfactory way of administering this substance.

Prognosis. This progressive muscular dystrophy of the usual type that I showed you first, with the proximal weakness, follows a course that is continuously and uninterruptedly downhill. Our patient is now worse than she was when I first saw her a couple of years ago. Two years hence she will be still worse. There is no chance as far as I know of her getting any better. Fortunately for her, the disease began unusually late in life and in those patients in whom the disease begins late the progress is apt to be slow. I have seen one man with this condition who was sixty-five years of age, and who still got around his home by crawling on the floor. The onset in his case had been relatively late, and the progression very slow. He was the oldest man I had ever seen with progressive muscular dystrophy. But generally speaking, these patients go downhill steadily and without interruption despite all treatment.

Here is a challenge for those of us who are interested in the chemistry of medicine. If we can solve these problems as to the chemistry of muscle physiology, perhaps we can learn how to help people suffering from a very common condition. It will certainly be welcomed by them.

The Charcot-Marie-Tooth type of progressive muscular dystrophy, as illustrated by our patient with the foot-drop, begins commonly in childhood, at eight to twelve years of age, progresses for some several years up to perhaps the age of maturity, and then commonly stops progressing. This boy's mother, still active, working, attending to her household duties, has the same disease. I don't remember her exact age but it must certainly be forty-five or fifty. Her condition is stationary at about the same degree of disability as her son's. She has reached her plateau and has remained at that level of disability. I believe this boy will do the same and will not become totally disabled as the first patient certainly will become.

The progressive muscular dystrophy in the second case, which is associated with attacks of family periodic paralysis, is so slight, and began so late (in fact he had never noticed it until we found it on examination) that I think he may escape ultimate disability. I have said enough about the treatment of his periodic attacks of paralysis to indicate to you that the prognosis in that regard, so far as we can see, is excellent.

DIAGNOSTIC SURGICAL CLINIC

Penetrating Injuries of the Chest

By RICHARD DAVISON, M. D.
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This subject of chest injuries is important at all times but particularly important in the light of present-day events. Needless to say, we have all types and all degrees of chest injuries. The type which merely consists of injury to the superficial structures and does not affect the underlying important viscera are of little interest. The same fundamental principles apply in the treatment of chest injuries as apply to other types of injuries. Further, there are special points pertinent to the vital functions in the chest, those of respiration and circulation.

I have chosen this morning a case that was admitted to the Cook County Hospital on the 25th of September, a seventeen-year-old boy who illustrates most of the important features in the special management of chest injuries. There is not a great deal visible for you see he is now apparently well and comfortable. I may say that almost without exception patients who do come to the hospital for treatment recover, many of them without any great difficulty. Others require the application of special principles of chest surgery.

This boy is seventeen years old with a history of being out on a party the evening before admission. He was stabbed three times. On admission the statement is recorded that he was sleepy. He doesn't know who stabbed him or just exactly how it happened. We find that the history is not made up for just the purpose of use in this clinic, but taken as is. In the handling of this case there were some mistakes made, but it is just as well to point out a few mistakes as well as to outline the proper course of events.

The first notation is that the pulse was 96 and blood pressure 112/80. That does not speak for any degree of shock. Of course, the first principle in the treatment of any injury is the management of the shock. I have been asked time and again: Is there a difference between shock in chest injuries and the shock in other injuries? The answer is essentially "no" and the same steps are to be taken in the management of this shock as in the management of ordinary shock. However, especially when there is open chest wound or a lax chest wall as a result of severe injury, there may be an additional factor in this shock, that is shifting or pressure on the mediastinum interfering with the mediastinal veins. However, again, the management of the shock is the same as in other cases, although in addition, any alteration of intra thoracic pressure should be corrected.

Three lacerations were found anteriorly, one in the first interspace, one right over the precordium, the third one over about the eleventh rib in the axillary line. Also there were some cuts about his face.

There was air sucking in and out of this major wound in the first interspace on the left side. In other words, we had a sucking wound, the result of an open communication from the outside to the pleural cavity, possibly also an open communication with the pleural cavity through the bronchus with air rushing into the chest because of the negative pressure into the chest. This results in the establishment of a pneumothorax, properly called a traumatic pneumothorax, but similar to a spontaneous pneumothorax.

There is a notation that he had blood clots on his lips, suggesting that he had expectorated blood and therefore the lung most probably was injured. That is an important point in the history.

The first step in any sucking wound is to close the opening in some manner or other. Usually there are available tissues. An open chest wall is not compatible with life in the ordinary individual for any period of time and this chest opening must be closed either with the tissues at hand, strapping, sutures, or with packing. In severe injuries with the wide-open chest the lung can be used to tamponade the opening. The chest wall, of course, was promptly closed. There is no notation in the history here as to whether or not he was dyspneic or cyanotic. The presence of dyspnea and cyanosis distinctly points to interference with the respiratory mechanism. The respirations may be quite rapid, in the state of shock, but I have never seen a patient dyspneic and cyanotic from shock alone. It means that something has happened in his chest to interfere with respiration. Almost always what has occurred is a collapse of the lung, possibly to a degree where even the other lung is encroached upon by shifting of the mediastinum. He apparently was not dyspneic or cyanotic.

The left chest was hyper-resonant, there were diminished breast sounds, there was crepitation around the wound, all pointing to pneumothorax with collapse of the lung. Some of the air may have come from the outside. More likely the air came through the tear in the visceral pleura. The crepitation in the wound occurs as a result of the positive pressure on expiration forcing air out in the tissue.

Emphysema is important here, primarily for diagnosis. In other cases of chest injuries, however, emphysema may go on to a point where the face is ballooned up. The body may be twice its normal size with extreme emphysema. There is quite a demand to do something. The usual question that is brought up to me is: "Isn't a rib or something sticking into his lung?" What can we do? People want something done. Knowing the mechanism of the development of emphysema, the treatment of the emphysema is very clear. It is due to air being pumped in the tissues, primarily on the expiratory phase at which time there is a positive pressure in the pleural cavity. The air must get into the pleural cavity in some way and its usual point of entrance is a tear in the visceral pleura. If air is pumped in by positive pressure, the obvious method of stopping this process is to put a permanent needle or a catheter into the pleural cavity and maintain constant controlled negative pressure in the pleural cavity. Then, no more emphysema can develop. In addition to this we may make multiple small puncture wounds to relieve the emphysema. The inhalation of pure oxygen may hasten the absorption of the emphysema. Have no fear of emphysema itself. If the patient is not dyspneic or cyanotic do not get alarmed. I know of no case which has developed an air embolism from emphysema.

What further was done for this patient? Obviously, again, the same points as far as the treatment of the superficial wound apply to this injury as to others: cleansing of the wound, débridement, removal of foreign bodies. The wounds were cleaned up thoroughly. Likewise the usual application of antitoxin, tetanus and polyvalent anaerobic serum was applied. We do not use chemotherapy routinely in these cases. Very few of them become infected and none was administered to this case.

Inasmuch as he presented no alarming symptoms of shock and no alarming symptoms of dyspnea and cyanosis, very little was necessary. One statement is that sulfathiazole was ordered by mouth for this patient a day or two after injury because the temperature went up to 101. It continued elevated for some time.

What was done, and what should be done about this spontaneous pneumothorax that develops frequently following almost any type of chest injury, particularly of course the penetrating injuries, such as gunshot wounds and stab wounds? Nothing was done here. The diagnosis of pneumothorax was made and yet nothing was done. Why? Because that was the proper course of procedure. Up to a certain point the development of a traumatic pneumothorax is a desirable therapeutic incident. This collapse of the lung helps in closing the opening in the lung. This collapse of the lung often aids in the control of bleeding from the lung. Therefore, as long as there is adequate oxygen exchange there is no necessity for interference. Some even encourage the continued maintenance of that pneumothorax. We merely leave these patients alone. However, if there had been dyspnea or cyanosis that would have been evidence that this traumatic pneumothorax had progressed beyond the desirable point and on to the point where interference was necessary. Usually if the collapse has developed to such a degree the heart is shifted to the opposite side. If such a condition does exist, dyspnea and cyanosis from a tension traumatic pneumothorax, the patient must be given relief, obviously, in the manner of simple reversal of the cause of his distress. The cause of his distress is air infiltrating into the pleural cavity. The method of giving relief is to take that air out. How are you going to take that air out and how much air are you going to take out? The simplest facilities at hand almost everywhere are a needle and a syringe, so we introduce a needle into the pleural cavity and remove air, usually just that amount necessary to make him comfortable from the respiratory aspect. We do not necessarily try to remove all of the air. When it is necessary to remove the air we usually remove about one liter.

Of course, there are other methods of taking the air out, such as with the pneumothorax machine, or with some type of available suction. Negative tension in the chest must be controlled and should seldom exceed 10 to 20 centimeters of water pressure. It is not wise to connect the pleural cavity to an uncontrolled suction. The result may be edema of the lung, or possibly interference with the circulation in the mediastinal veins, so use only a moderate suction.

In the average case that comes into the Cook County Hospital that requires relief from this traumatic pneumothorax, we take air out once, usually a nominal amount of 500 to 1,000 cc. and from this point on the patient is comfortable. That is all that is necessary. I would estimate that in almost 90 per cent of cases that come to the Cook County Hospital no interference is necessary because the traumatic pneumothorax has not gone beyond the favorable point. In those cases where it is necessary to remove air, it is usually necessary to remove air only once. However, if the opening in the lung remains patent and air continues to filter into that pleural space, it may be necessary to remove air more than once, repeatedly, or if the opening is large, even continually.

For continued removal, we connect this pleural space with the aid of a needle or a catheter to some form of controlled suction. A simple Wangensteen suction may be satisfactory, although this may require repeated filling of the bottles if there is a continued leakage. We can set up a more complicated and possibly slightly better method of suction.

In this particular patient no interference was necessary. These remarks cover the management of traumatic pneumothorax which must be recognized and in certain cases properly combated, although I repeat that in most cases the pneumothorax requires little attention.

The next important special consideration in the management of chest injuries is the question of intrathoracic bleeding. Needless to say, any superficial or visible bleeding is always controlled, but if there is continued bleeding into the pleural space the patient may bleed to death silently. In all chest injuries we must be on a careful lookout for continued bleeding into the pleural cavity. In the few cases admitted to the Cook County Hospital who have not survived treatment, the most frequent cause of death has been continued intrathoracic bleeding.

This patient did not have the picture of hemorrhage when he came into the hospital. His blood pressure was normal. The next step was to follow this blood pressure at intervals of every hour or two for the first day. Evidence of continued intrathoracic bleeding would be a gradual increase in the pulse rate, a gradual fall of the blood pressure and changes in the blood count.

He was watched faithfully and there was no alteration of pulse or blood pressure and therefore no apparent continued intrathoracic bleeding. But suppose there had been, what measures would we institute? What should be done? For diagnosis we could insert a needle into the chest. The recovery of blood is evidence that there has been bleeding but not evidence of continued bleeding.

If there is continued bleeding, what vessel is that bleeding coming from? The answer is almost always the intercostal vessels, occasionally the internal mammary, but possibly some other small vessel. Obviously the great vessels are not involved because if these structures are ruptured the patient does not come to therapeutic consideration.

The first thought might be that the bleeding is from the lung, but as a matter of fact it is almost never from the lung. It is true that the lung is a vascular organ but the blood pressure in the pulmonary circuit is only one-sixth that of the systemic circuit, and, further, the collapse of the lung aids in controlling bleeding, so for practical purposes we exclude the lung as a source of continued bleeding.

Ransom, who did considerable work in the Chinese-Japanese wars, makes the statement that the lung need never be considered as the source of bleeding.

If there is continued bleeding and we must do something about it, what is there to do? At the County Hospital we merely write a check on the blood bank and replace the lost blood. Almost always single or repeated blood transfusions result in control of bleeding. How or why may be difficult to convincingly explain.

In this patient the exact site of the injury which penetrated the chest is known and this gives us a clue as to which intercostal vessel is involved. It would be possible either to enlarge this opening and tie off the intercostal vessels, or we could simply whip a suture around the rib anterior and posterior to the injury, thereby incarcerating the intercostal vessel. This simple expedient can be used, but is usually unnecessary.

In almost all cases of continued intrathoracic bleeding that we have at the County Hospital, simple replacement of the blood, usually no more than two or three times, results in control of bleeding and no desperate efforts are necessary.

There is a demand if the patient has persistent lung opening or if he is bleeding in the chest to open his chest, to suture the lung, to find the bleeding point. We find in the literature those who encourage frequent surgical intervention in cases of injuries of this type. In my experience, such interference is almost never necessary. The patients recover, and such heroic methods merely add, I believe, to mortality.

It is not as easy as one might think to locate the bleeding point or even the opening in the lung. Reports from the war areas concur in this viewpoint, only a small percentage of cases being subjected to any surgical intervention other than in treatment of the wound or drainage of the infection.

These two points, the management of traumatic pneumothorax and management of intrathoracic bleeding, are the two most important special points in the management of chest injuries.

There are, however, one or two other factors. One is the management of the residual hemothorax. About 70 per cent of all chest injuries have a hemothorax. This blood does not clot readily and may remain liquid for days, weeks, or even months. Why? It is explained that the blood becomes defibrinated and the pleural cavity may have a mesothelial-like action similar to blood vessels. There is a school of thought which advises merely leaving the blood alone unless there is enough blood in the thorax to cause pressure symptoms. If such a quantity is present it must be removed as an emergency measure.

Disposition of this question is left until the patient's condition is improved, usually several days after the accident.

In my opinion, small amounts of blood rising up to the tenth rib require no intervention. If the accumulation rises to the eighth rib, the blood should be aspirated and aspirations repeated. The blood may eventually clot, clots contract, calcify, and the result may be considerable pain, disability, deformity, and encroachment upon good lung tissue. What is more important, the blood constitutes a suitable culture media for infection. If it is removed there is less likelihood of the development of empyema which may not be particularly serious but is at least a distressing complication.

This patient's hemothorax was aspirated and significant amounts of blood were withdrawn. The end result has been a complete clinical recovery, the only residue being a homogeneous opacity which is due to thickening of the pleura. The lung has now re-expanded.

Of course, any structure in the chest may be injured. The diaphragm may be penetrated, and we may encounter combined intrathoracic injuries. Time does not permit discussion of other types of chest injuries.

In conclusion I will repeat: The same principles in the treatment of other wounds apply to the treatment of chest injuries. In addition, we have these special points: recognition and management of traumatic pneumothorax, recognition and management of intrathoracic bleeding, the disposition of the residual blood, and treatment of complicating infections.

DIAGNOSTIC MEDICAL CLINIC

Obesity in Children

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It is a great pleasure to be with the International Medical Assembly again. I am sure I can speak for the medical profession of Chicago when I say that we are delighted to have you meet in Chicago once more.

The title of the clinic today was not one selected by me but was one assigned to me, Obesity in Children. Many interesting problems are raised by this title, such as the mechanism of obesity and the definition of obesity. We could discuss various phases of this problem at some length, but time is short and we have several patients to show, so I am going to limit my remarks primarily to a discussion of dystrophia adiposogenitalis, which in children is called Fröhlich's syndrome.

One point that I want to make is that an endocrine disorder in childhood is not necessarily associated with obesity. For example, (slide) here is a cretin twenty-one years old, who is extremely short and about the size of a normal child three or four years old. She is not obese and she is not developed sexually.

Will you stand up, Johnny? You see here a thirteen-year-old boy who is a pituitary dwarf. As a matter of fact, only a short time ago, before he was treated, he was thirty-eight inches tall. He is now forty-six inches tall. He had grown very little over a period of many years. The treatment consisted in the administration of chorionic gonadotropin which stimulated the testicles to produce male sex hormone and, as you know, male sex hormone is one of the most potent stimulators of growth available at the present time. The main thing I want to point out to you is that we may have extreme hypopituitarism without any obesity. It may, of course, be associated with obesity, and whether it is or not may depend upon factors outside of the pituitary, notably upon an appetite center in the hypothalamic area just above the pituitary. As you know, the hypothalamic area can be punctured in animals without injuring the pituitary at all and such animals very promptly develop an enormous appetite and gain a tremendous amount of weight but their pituitary functions may go on undisturbed.

There are many reasons, therefore, for believing that in patients with hypogonadism secondary to hypopituitarism in whom obesity is present, the obesity may be the result of an associated involvement of the floor of the brain just above the pituitary, notably in the hypothalamic area which is the site of many important functions.

(Slide) Here is a young woman with marked hypothyroidism, perhaps secondary to hypopituitarism, who at the time this picture was taken was thirteen years old. I had hoped to have this patient present this morning, but she was not able to come. In association with the administration of thyroid she has matured sexually. Her body contour has changed completely; she is a perfectly normal looking young woman with menstrual periods at regular intervals. You notice that she was not terribly obese before treatment. She was somewhat. In other words, hypothyroidism per se is not a cause of obesity.

Here is a young woman eighteen years old, very short in stature, only about fifty-three inches tall. She had never menstruated. She had hair on her forearms and legs, calcification in the region of the adrenals, and we had every reason to believe that she had an over-production of androgenic material by the adrenal cortex early in life, as a result of which there had been premature closure of the epiphyses. She had failed to mature sexually and did so only when we gave her large doses of female sex hormone.

(Slide) However, hypogonadism may be associated with obesity, as you see in this particular instance of a sixty-year-old man who had had undescended testes, both of which were removed at the age of nineteen. Within one year following their removal he developed large breasts and gained 100 pounds. The combination of obesity and hypogonadism is rather common but the two do not necessarily go together.

(Slide) Here you see a eunuchoid individual. He is nineteen years old, normal in height, sixty-seven inches tall. Once again I wish to impress upon you the fact that he is not obese. When he received male sex hormone and sexual maturity was thereby inducted, he actually gained forty pounds in four months, but he did not become obese. You note that the weight is well distributed in the photograph on your right; his muscles are much better developed than before treatment, and he is a much more mature looking individual.

(Slide) This young woman actually gained twenty pounds when female sex hormone was used to induce sexual maturity. She had not menstruated, had flat breasts, an infantile uterus, and so forth, at the age of eighteen. During the administration of female sex hormone she actually deposited fat but became much more pleasing in appearance because it was deposited in the characteristic feminine areas and, of course, the size of her breasts increased and other changes set in.

(Slide) Just to show you that marked hypopituitarism in the first patient does not necessarily bear any relation to pituitary function, here is a man with a chromophobe adenoma of the pituitary who, if anything, is less than normal in weight.

(Slide) A similar phenomenon is observed in this man who developed Simmonds' disease, extreme hypopituitarism with loss of all pituitary functions, following a severe attack of influenza. His metabolism dropped but he was not obese; he actually lost some weight.

(Slide) I just want to say that much to point out to you that a pituitary deficiency is not necessarily associated with obesity. Now we come to the subject which concerns us principally today, namely, that of the Fröhlich's syndrome, an example of which you see here in a six-year-old boy. It is very difficult for me to believe, as some pediatricians would have us believe, that this individual is normal. You have seen young boys and young girls like this one. You note that his breasts are full, his abdomen large, and he has knock knees. You note the characteristic transverse fold in the lower part of the abdomen just above the pubic area.

There are all degrees of this condition. The amount of hypogonadism associated with it is variable. We see its counterpart later in life, as I shall show you.

(Slide) Here is an untreated nineteen-year-old boy. He has shown some attempt at sexual maturity, but it is only partial and he still is typically Fröhlich in type. He has the short trunk and long extremities of the eunuchoid individual, large abdomen, and so forth.

(Slide) A thirty-seven-year-old man weighing 325 pounds with one undescended testis who is characteristically of the same type.

(Slide) A woman with a similar disturbance weighing 375 pounds.

A pituitary tumor is rarely found in individuals who have this condition in marked form. The sella turcica is almost invariably normal in size. In the case originally described by Fröhlich in a young boy there was a cranio-pharyngioma which compressed the pituitary and thereby interfered with its function. The boy was dwarfed because the production of growth factor was interfered with. Most of the individuals of the Fröhlich type that we see are normal in height because there is usually no pituitary tumor and a normal amount of growth factor is produced. If a chromophobe adenoma is present there may be associated dwarfism.

(Slide) A similar disturbance in another woman.

I should now like to show you a boy who less than a year ago was of the type that I have been discussing. This boy was typically Fröhlich in type. I think you would agree that now he is a perfectly normal looking boy. How old are you, Bennett?

Patient: I will be fourteen in March.

Dr. Thompson: He is thirteen and a half years old. Do you feel any better since you have been treated?

Patient: Yes. I don't get tired out as fast. I can do a lot more work and everything.

Dr. Thompson: I think his mother can tell the story a little better. What change do you notice in your son?

Mrs. N.: A great change.

Dr. Thompson: What change, would you say?

Mrs. N.: In all ways he is more active.

Dr. Thompson: What else do you notice? Has he more interest in playing games?

Mrs. N.: Yes, he wants to go out with the other boys more. He used to play around the house and didn't want to go out at all.

Dr. Thompson: He used to like to play around the house, sit down and read all day, is that right?

Mrs. N.: Yes. He is a hundred per cent better.

Dr. Thompson: Now he is a perfectly normal boy, "a hundred per cent better." The treatment that this boy has received consists of two things: (1) He has been given material which stimulates the function of his testicles, namely, chorionic gonadotropin, which I have talked about to this group before. It is the gonadotropic material prepared from the urine of pregnant women. He has received this material in a dose of 500 international units three times a week since about the first of December of last year. In association with the administration of this material he has been given a weight reducing diet. He does not stick to this completely. His mother tells me that periodically she feels sorry for him and gives him some cake and pie. Is that right?

MRS. N.: Once in a while.

DR. THOMPSON: As a matter of fact, the changes which you see in this boy may occur without the administration of a weight reducing diet at all.

(Slide) Here is a picture of that boy last November before he was treated. I think you will agree that he has been completely transformed. Even his knock knees have disappeared. We began treatment at the age of twelve. I would say that it would be desirable to begin treatment just a little earlier, preferably between the ninth and eleventh years. You must begin treatment just before the age at which puberty sets in and it must be continued until the epiphyses unite completely in order to influence as much as possible the configuration of the skeleton, which, as you know, bears a very important relationship to the production of sex hormones.

(Slide) Here is another young boy of the same type, only in this instance much younger. I think I am correct in saying he is seven years old. We began treatment at the age of six years. This boy was very obese and had all the characteristic manifestations that we have been describing. He is much younger than most of the boys that I shall show you. He showed exactly the same changes that we have described in the preceding patient and with the same kind of treatment. After some increase in the size of his genitalia had taken place it was felt that it would be wise to discontinue treatment, and when this was done his appetite increased and he has gained a considerable amount of weight and is beginning to show many of the changes that were present before any treatment was started.

The problem, of course, comes up as to what age treatment should start in individuals of this type. The ideal age probably is between the ages of nine and eleven years, although something should be done at an earlier age because some of these individuals become extremely obese. It may be that the best form of treatment will eventually work out to be periodic administration of chorionic gonadotropin at an earlier age with careful supervision of the diet of these individuals.

Here is another boy who presents in addition to the changes described some deficiency in the function of the cerebral cortex, not terribly great, but just enough to retard things a little. He has all of the other changes that we have described. He has been under treatment now for about two months and has shown very definite improvement. I think his father can tell you the change which has occurred.

MR. S.: Very definitely Herb has improved. We had an I. Q. test taken of him some time ago. It seems that since he has had these shots for the last two months he has perked up mentally and seems to be very much more self-assertive than he was in the past. He had in the past received other types of treatment that didn't seem to help as much as Dr. Thompson's. Is that what you wanted, Doctor? (Laughter)

(Dr. Pfeiffenberger accidentally tripped on a cord in walking past the speaker and broke the glass on the desk of the rostrum.)

DR. THOMPSON: I got a little suspicious when Dr. Pfeiffenberger walked over there again. You will be interested to know what he said to me as he passed. He said, "You've got somebody else telling stories for you." (Laughter)

Here is another boy who has been completely transformed. Will you stand over at the microphone? About a year ago he was just like the boys that I have shown you pictures of. Do you feel any better?

PATIENT: Yes, sir.

DR. THOMPSON: In what way?

PATIENT: Well, before, I felt generally run down. Now I feel in the best of health.

DR. THOMPSON: What he means to say, I presume, is that he fells like a normal boy and wants to do everything at the present time. Is that right?

PATIENT: Yes, sir.

DR. THOMPSON: Formerly he was terribly sluggish and his mother felt very much worried about him.

(Slide) I will show you this patient in a few minutes. We have pictures of him in various stages of development from the age of about seven or eight years up to the present time when he is fifteen. He did not begin treatment until about two years ago and his treatment has been very intermittent. He has been given treatment with male sex hormone instead of chorionic gonadotropin. In other words, we have given him substitution therapy. There has been a definite increase in size of the genitalia but the changes are not as great as we would like to have seen them, the reason being that he has been very spasmodic about coming to the clinic.

Here is another boy in whom there has been a very striking change. Here is a picture of the little boy you saw, the seven-year-old boy, before treatment and after a few months of treatment (Slide).

I just want to point out to you that the end result of failure to treat young fat boys is not necessarily dystrophia adiposogenitalis in later life. Here is a young boy who appeared to have this condition in a mild form and we decided to use him as a normal control, and without any treatment at all he developed into a normal boy. There is a period of four and a half years between these photographs and he did not grow nearly as fast as the boys who received treatment grew, but there has been, without treatment, a very striking change. It is very important to know when to treat these patients and when not to treat them, and it is not an easy problem to decide.

(Slide) In contrast to the effect of treatment, here is another boy sixteen years old in the photograph on your left, and nineteen in that on your right. He was not treated. He has lost some weight, the reason for that being not sexual maturity, as you can see, but the development of rheumatic fever. You note that all the characteristic changes of the Fröhlich syndrome have persisted and there has been no attempt at spontaneous change.

(Slide) Here is a boy nineteen years old with all the characteristic changes we have described but no pituitary tumor. We have been able to make him grow four inches but we have not been able to change his body contour as much as we would have liked, the reason being that the treatment was started too late because he did not come to the clinic soon enough. It is very important to begin treatment early enough.

(Slide) Here is a similar type of change in a young girl ten years old, (slide) and on the left of the next slide is her mother before treatment, in the center after she had lost some weight, and on the right after she had gone on an eating spree again.

(Slide) Here is a boy similar to the one on the stage. You note the very striking change that occurred in him with chorionic gonadotropin alone.

(Slide) In the next slide you see another boy in whom treatment was started a little late. He was sixteen years old when treatment was started, so the growth capacity of the skeleton was very limited. It should have been started much earlier.

(Slide) Here you see the patient who is with us now, before and after the administration of male sex hormone, and here is the patient himself, a perfectly normal looking boy. He is not as old as you probably think he is. How old are you, Richard?

PATIENT: Thirteen.

DR. THOMPSON: You notice that although he has been treated and his epiphyses have begun to close a little early, he is not short. He is perfectly normal in height, as a matter of fact, he is five feet nine inches tall. Do you feel any better since you have been treated?

PATIENT: Yes.

DR. THOMPSON: In what way do you feel better?

PATIENT: Well, I can get around better.

DR. THOMPSON: Do you play on the football team?

PATIENT: Yes.

DR. THOMPSON: What grade are you in in school?

PATIENT: 1-A in high school.

DR. THOMPSON: He plays on the high school football team. Do you play on the first team?

PATIENT: I am a substitute at present.

DR. THOMPSON: He will be in the first team very shortly.* Without treatment I don't think he would be.

Here again is a patient who should have started treatment somewhat earlier. He is sixteen years old at the present time. The width of his hips and his body configuration are somewhat fixed by the fact that treatment was begun rather late. A great deal can be done for boys as long as their epiphyses remain open, but once they are closed, of course it is not possible to do anything.

The next patient that I will show you is a boy of the same type who had one undescended testis which has come down with glandular therapy. Do you feel any better, John?

PATIENT: A little.

DR. THOMPSON: I should have shown him first. (Laughter) Treatment has just been started. (Laughter) You may go. (Laughter)

I now wish to show you four patients who illustrate various other phases of this problem. On your left you see a boy with the Fröhlich syndrome who had a slipping of an epiphysis in one knee to a very marked degree. He is under treatment but treatment was begun rather late. However, definite changes are occurring.

The boy next to him has bilateral intra-abdominal testes, in spite of which we have been able to produce a very definite change with chorionic gonadotropin alone, with stimulation of growth. We could have done much more if the testes had been in the scrotum and more capable of responding to stimulation.

Next to him you see a boy who is untreated in whom the characteristics of the Fröhlich syndrome are persistent at the age of sixteen.

Next to him you see a boy who is under treatment with methyl testosterone by mouth in whom very striking changes are occurring. He still is a little obese, but his body contour is undergoing very rapid modification.

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*He made the first team before the end of the 1943 season.

So much for the patients. I want to say just one word in closing. When you are faced with the problem of a young boy of the type we have described, with marked hypogonadism, what should you do? When should he be treated and when should he not be? There is no absolute answer to that problem at the present time. I think it is fair to say that if the manifestations of the Fröhlich syndrome are marked and hypogonadism is marked, it is wise to carry out treatment at the age of nine to eleven years with chorionic gonadotropin given in a dose of 500 international units three times a week. This treatment should be continued until epiphyseal closure sets in. If the individual fails to lose weight in his abdomen and hips at a satisfactory rate, this treatment should be augmented by a suitable weight reducing diet which in most instances should contain about 1500 calories. If there remains a very definite depression of the metabolism, these two forms of treatment should be supplemented with the administration of desiccated thyroid.

One further word. There has just appeared in a leading magazine a summary of Newburgh's work to the effect that all obesity is the result of overeating. Newburgh has re-emphasized a very important point, namely, that the law of the conservation of energy applies to biologic phenomena. However, from the facts I have presented to you and from the patients I have shown, I think we would be forced to conclude that there is more to overweight than overeating.

DIAGNOSTIC SURGICAL CLINIC

The Importance of the Surgical Anatomy of the Abdominal Wall with Special Reference to Abdominal Incisions

By ALBERT O. SINGLETON, M. D.
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Since I have been placed in the diagnostic clinic group and I have no patients, I should like to reverse the situation and use the surgeons of the audience for my patients and see if I cannot diagnose some of their ills.

Those of us who have been rather closely associated with medical education for a long span of years and have seen anatomy a major subject in the fundamental subjects and now find it to be a minor subject, become more or less disturbed about the teaching of anatomy and I feel that if there ever was a time for surgical anatomy to be emphasized it is at this time when didactic anatomy in medical schools is being given such a very small place in the curriculum.

What I have to say will be directed to those surgeons who, first, have at one time or another had difficulty in getting a good exposure of the field of operation in certain abdominal operations; to those who occasionally have wound disruptions occurring postoperatively; to those who have had their patients or other surgeons' patients come to them with postoperative hernias; to those whose patients may have painful postoperative convalescence or too frequent pulmonary complications; to those surgeons who have a fear of wound failure and use some special means of preventing it, such as special suture material of chromic catgut, silk, cotton, or wire, or special sutures, such as figure of eight or deep sutures, or special supports of the abdominal wall postoperatively, such as adhesive strapping or abdominal binders.

The long vertical incisions (midline, right rectus, left rectus) were at one time probably justified when intraabdominal diagnosis was less precise, but with modern diagnosis and diagnostic agents one is rarely required to do an exploratory operation. The age-old incisions are so firmly fixed in the habits of surgeons that it is difficult to get away from them. Not only are these incisions faulty anatomically, but often the best exposure of the operative field cannot be secured through them. The average surgeon is so intent upon getting to the pathology in question, the stomach or the gallbladder, the diaphragm or the colon, that he resents the delay caused by the comprehensive knowledge of the abdominal wall, though he will study and know minutely every detail of the technic for removing the organ in question.

It is true that the contributing factors to faulty wound healing are many, but in our opinion a proper regard for anatomic incisions makes the other causes assume a place of small importance. It is evident that an unsatisfactory restoration of the abdominal wall is quite common when one investigates the literature and our experience is certainly convincing.

We have made a careful study of 9,000 consecutive abdominal incisions in our institution, the John Sealy Hospital at Galveston, and this furnishes sufficient evidence of the truth of the above statements.

TABLE I.
Review of 9,000 Consecutive Abdominal Incisions for Disruption and Hernia

				Disrupted		Hernia	
Incision	No.	Clean	Drained	No.	%	No.	%
Nonanatomic							
Midline	4,464	3,715	749	36	0.80	87	1.93
Lower rectus	1,097	881	216	15	1.37	28	2.55
Upper rectus	292	226	66	9	3.08	16	5.47
Total	5,853	4,822	1,031	60	1.02	131	2.24
Anatomic							
Pfannenstiel	29	29	0	0	0.0	0	0.0
McBurney	2,648	1,987	661	1	0.037	28	1.05
Modified Sloan	382	278	104	0	0.0	1*	0.26
Lateral transverse	88	68	20	0	0.0	0	0.0
Total	3,147	2,362	785	1	0.031	29	0.92

*Omentopexy.

(Slide) In this review of 9,000 consecutive abdominal operations, the first group of patients, 5,853, have been classed as non-anatomic abdominal incisions. Of the 5,853, midline, lower rectus, upper rectus, the disruptions of the wound were 60, or 1.02 per cent; postoperative herniae which we were able to trace were 131, or 2.24 per cent. Probably this would have been more than doubled if we had been able to trace all of the patients.

Of what we have classed as anatomic incisions, the Pfannenstiel, McBurney, modified Sloan and lateral transverse incisions, 3,147, there was one disruption, or 0.031 per cent, and 29 hernias, or 0.92 per cent.

This in itself would indicate that we are justified in claiming that the anatomical incisions are well worth while and that the old vertical incisions which we are so in the habit of using can be dispensed with.

The important structures of the abdominal wall, of course, are blood vessels, nerves, muscles, fascia and skin. Fortunately, the abdominal wall is richly supplied with blood from many sources and the sacrificing of certain arteries may be done with impunity. One rarely is afraid of devitalizing the abdominal wall sufficiently to prevent union by making incisions anywhere. The intercostals, superior and inferior epigastrics, with their many anastomoses, supply the abdominal wall freely with blood. Also, the nerve supply, both sensory and motor, is abundant. Because of the many intercostal nerves and their intercommunications, it is difficult to denervate any part of the abdominal wall, though numerous nerve trunks may be severed.

(Slide) This is a very familiar anatomical dissection showing the nerves of which we have spoken. It is possible to cut one, two or three of these intercostal nerves and not denervate the rectus muscle; neither will it denervate the flat muscles or the muscles which are lateral to the rectus. We have tested patients who have had these incisions with nerves cut and invariably within two months' time the restoration of sensation and muscular power was complete.

The rectus muscles are the only muscles that run parallel with the length of the body and these muscles are largely used to splint the abdomen and protect the abdominal structure. The other muscles are, of course, the flat muscles, as we call them, the internal oblique, the transversus and external oblique. These muscles have a function which is much more complicated than the rectus abdominus. These muscles not only maintain the positive pressure in the abdomen—they almost encircle the body—but at the same time they are respiratory muscles and are never at rest, and during extra effort such as retching or vomiting or sneezing, the pull on these flat muscles across the abdomen is very great. The muscle fibers run in a transverse direction; therefore, in order to maintain a satisfactory abdominal wall, incisions through them should be in a transverse direction.

The fascia of the abdomen is the most important holding material we have and the fascia must be always thought of as the tendinous continuation of the muscles, the fascia in the upper abdomen particularly. Here we have another familiar diaphragm showing the flat muscles, laterals to the rectus muscles and their continuation, the fascia in front and behind the rectus muscles which forms the linea alba. These are nothing more than the tendinous continuation of the muscles that are lateral to the midline. The fibers of these muscles as well as the fascial strand run in a transverse direction. If one cuts this fascia vertically, it requires a tremendous amount of effort to maintain it in position afterwards, particularly during effort such as sneezing, coughing, or gaseous distention.

The controversy over suture material is largely due to the fact that surgeons endeavor by the aid of suture material to overcome this tremendous pull, and the controversy over the use of various types of suture material will continue without a decision as long as surgeons disregard the anatomical arrangement of these structures and try to restore the abdominal wall after unanatomic incisions.

There is hardly a place in the abdomen where one cannot secure adequate exposure for surgical operation and still make the incision between the muscle fibers or fascia fibers parallel with the line of tension and thus avoid the difficult problem of keeping the wound edges together. If the tension on the wound is great enough to require strong sutures, the result will be strangulation of the wound edges to such a degree that union of the tissues will be greatly interfered with; while on the other hand if an incision is made in such a way that the edges will be approximated without suture tension, the tendency to disrupt is not present and good circulation will prevail and union will be assured.

The greatest difficulty is encountered in the upper abdomen and here particularly are we able to make the transverse and modified transverse incisions with assurance of restoration of the abdominal wall much more satisfactorily than with the vertical incisions. A few surgeons have been interested in this problem over many years. Transverse incisions of various types have been advocated by Pfannenstiel, Boeckman, Moskowitz, Quain, Bartlett, Sloan and others. Some of these, such as Moskowitz, and recently

Rees and Coller, have advised the transverse incision as a general principle, disregarding anatomical structures to the extent that the recti muscles are cut across. Gurd has also concurred in this general principle. While such incisions are sutured with much less tension upon them than the vertical incisions, still one can hardly justify the cutting of the rectus muscle when it can be preserved with perfect ease and still serve an important purpose during healing and afterwards.

Every surgeon should have a working knowledge of the anatomy of the abdominal wall, and with this knowledge he may open the abdomen at any location with a safe muscle and fascial splitting incision, giving good exposure and a sure approximation of the tissues involved.

There are many instances in which a vertical incision does not allow the surgeon to have access to the organ which he is trying to reach. When we consider making vertical incisions for operations involving the ascending colon, the cecum, or the gallbladder, which is often very far lateral to the midline, also the incision in the left half of the colon, and an effort to remove the spleen through a vertical incision is also a hazardous procedure, one is better off frequently, as far as getting access to the field of operation is concerned, by avoiding the vertical incision and making incisions lateral to the midline.

The first one of the splitting incisions which became popular many years ago was, of course, the McBurney incision. Many surgeons avoid the McBurney incision, chiefly because it does not give them room to work very well and to explore. A McBurney incision, in the opinion of most of us, is the incision preferable for operations upon the appendix, but one must be in a position to make the McBurney incision in its typical form and modify it so it can be used, if one wants more room, without endangering the abdominal wall. The McBurney incision can always be enlarged upward or downward by incising the rectus sheath beneath the external fascia and secure more room, the pelvis may be reached, operations upon the uterine adnexa done, even resection of the right half of the colon can be done, if one makes a McBurney incision and finds that he has some other complication or some other diagnosis which he did not anticipate.

(Slide) This patient is a young girl nineteen years of age who had had a chronic ulcerative colitis since childhood and she developed a tender area in the right side. She was seen some several weeks later and it was questionable whether she had had an appendiceal abscess, a perforation of the cecum from ulcerative colitis, or granulomatous growth of the cecum, or possibly whether she had a malignancy. Her age made us feel that possibly the last thing we wanted to believe was a malignancy. A high McBurney incision was made and exploration revealed a carcinoma of the ascending colon. The incision was enlarged very easily, as demonstrated in the previous diagram, the right half of the colon resected, leaving a wound that is perfectly substantial in every way and giving a very good access to the field of operation, even better than the right rectus incision which is customarily used.

(Slide) The upper abdomen, as we have said before, is the location in which most disruptions occur and in which there is greater tension than in other parts of the abdomen, in which there is a greater amount of fascia, and here one may make anatomic incisions without the fear of disruptions and wound separation and at the same time get excellent results.

The Sloan incision is the most perfect of all the anatomical incisions, I think, from the standpoint of anatomy, and it is available and a working knowledge of it should be had by every surgeon, because in that way he will certainly become familiar with the anatomy of the abdominal wall from the incision standpoint.

The skin incision may be made transversely or it may be made vertically. A little more dissection is required in the transverse incision. The anterior rectus sheath is incised and the rectus muscle dissected, the anterior sheath dissected off the rectus muscle to the midline, and then the rectus sheath below that. The incision is extended across the linea alba, across the anterior portion of the rectus sheath on this side, and then the rectus muscle pulled this way, retracted that way, and here we have a demonstration of the tremendous amount of support the abdomen is given by this strong fascia, all pulled in this direction. The incision may be enlarged very easily and exposure gotten for almost anything one wants to do in this part of the abdomen.

If resection of the stomach is high, it is necessary that the rectus on both sides be cut and pulled to the side.

When the wound is brought together the pull is in a transverse direction. The more the patient strains or retches or tightens the flat muscles, the more it pulls this incision together. There is no tension; it does not matter what kind of suture you put it together with, it can never come open once the material is sutured.

The scar that you make by transverse incision is naturally less noticeable than the scar of vertical incision, though this is a very minor point.

The lines of cleavage of the skin, as we know, run transversely. The tension upon the skin in the vertical direction is not very great but it is great in the lateral direction, but again the scar is something of very minor importance.

(Slide) We should also keep in mind that in pyloric stenosis in infants, in whom the abdominal pressure is very great, by a muscle-splitting incision to the right, much farther to the right in the infant than in the adult, one is able to bring the knuckle of obstruction out of the wound, and incise it without the escape of the intestine round about. The same is true of gastrostomy on the other side: a muscle-splitting incision, cutting the posterior sheath transversely by all means, and here again there is no tendency for this wound ever to come open and give subsequent trouble.

In the right side of the upper abdomen an incision we have used and others have used in some form we call a lateral transverse incision, which gives one access to the colon on this side, the hepatic flexure of the colon particularly, but more particularly the gallbladder and biliary passage. This incision is made parallel with the costal border an inch or two below; the anterior sheath of the rectus muscle, which is a continuation of the external oblique fascia, is cut across, and then the skin incision will be seen to be in a line with the internal oblique, which is the strongest of these flat muscles. The rectus muscle is retracted medially after the anterior sheath has been dissected off, and the posterior sheath cut beneath the rectus muscle and the incision extended down laterally to just posterior to the anterior superior spine, and between the fibers of the internal oblique.

This gives an approach to the biliary ducts and gallbladder that no other incision gives, and if it were not valuable for anything else I think this justifies the incision. With this incision the exposure one gets allows the difficult operations on these organs to be done without accident. Most of the accidents which occur to the biliary ducts during surgical procedures are largely due to the fact that the surgeon does not have good access to the area, and cannot see what he is doing, but with this incision one does see everything that is to be done. This incision may be made a little lower and has been used very satisfactorily for resection of the colon on the right side, or the middle portion of the colon.

The incision is closed, again there is no tension on the wound, the suture material does not have to be strong, and the wound tends to stay together. One row of stitches includes the posterior sheath of the rectus, internal oblique and transversus and the peritoneum, and the second row takes the anterior sheath of the rectus with the external oblique fascia. We can highly recommend this incision. On the opposite side it is just as useful for procedures on the colon or it can be extended further up in the epigastrium and still give the same results, where one may operate upon diaphragmatic hernia with much more satisfaction than with a vertical incision. It is particularly useful for the splenectomy. I have a great deal of respect for large spleens and have seen some very serious accidents from hemorrhage. This incision gives access to the spleen that no other incision will give.

The incision is made just as it is on the other side for the gallbladder. The splenic artery is ligated preferably first, the gastrosplenic omentum is tied all the way to the diaphragm before the spleen is disturbed, and then with the circulation completely controlled the spleen is delivered and removed with perfect safety. The splenic bed may be seen and dried after the spleen is removed so that no danger of leaving oozing surfaces exists.

TABLE II.

REVIEW OF 762 INCISIONS OF THE UPPER PART OF THE ABDOMEN

		Disruption		Hernia	
	No.	No.	%	No.	%
Vertical Incisions . .	292	9	3.08	16	5.47
Transverse Incisions	470	0	0.00	1*	0.21

*Omentopexy.

In a review of 762 consecutive upper abdominal incisions, vertical incisions were 292 with 9 disruptions or 3.08 per cent; herniae 16, or 5.47 per cent; transverse incisions, the ones we have been describing, 470, no disruptions, one postoperative hernia that we have been able to find. This comparison, I think, is convincing as to the value of these incisions.

SUMMARY

Surgeons should get away from the old vertical incisions, especially in the upper abdomen. Exploratory incisions are rarely necessary at the present time. By becoming familiar with the anatomy of the abdominal wall muscle and fascial splitting incisions can, in the great majority of abdominal operations, be used with better access to the field of operation. Disruption of wounds and postoperative hernia may largely be eliminated by avoiding unanatomical incisions. Postoperative discomfort is lessened and postoperative pulmonary complications reduced by using transverse incisions in the upper abdomen.

DIAGNOSTIC SURGICAL CLINIC

Obstructing Lesions of the Common Duct

By VERNE C. HUNT, M. D.
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Off the record, I might say that Chicago is devoid of cases of obstructive jaundice so we have no cases to present this morning, which I think is perfectly all right because you all have seen jaundice and I think the clinical cases would exhibit nothing more to you from this distance at least than the picture of icterus.

First of all, I think we should speak of the types of jaundice that are encountered clinically. While we might go into detail, we shall not do so, but for practical purposes we shall say that jaundice consists primarily of three types: the hemolytic type of jaundice, the hepatogenous type and the obstructive type.

Not infrequently considerable difficulty is experienced in differentiating the so-called hepatogenous or intrahepatic type of jaundice from true obstructive jaundice. In order that surgeons may avoid surgical exploration on patients in whom surgery is of no avail and accomplishes nothing, and may confine their activity to obstructive jaundice only, I think it is very important that we have pretty definite ideas regarding the various laboratory aids and clinical data which are of value in differentiating one type of jaundice from another. There are many refinements in the differential diagnosis of these various types of jaundice, but for practical purposes I think it really gets down to only a very few matters.

When I stated that one should have pretty definitely organized ideas regarding the value of laboratory aids and clinical data, I really meant to say that one should resolve to put first values first. At times clinicians and others have attributed too much importance to certain laboratory data and through confused interpretation unduly prolonged observation and study of some of these jaundiced patients has resulted. I have upon occasion stated that many patients with the various types of jaundice have been observed in the hospital, in wards particularly, for prolonged periods of time and all sorts of laboratory data have been accumulated which lead to continued, repeated and prolonged differential diagnostic seances without any particular benefit to the patient.

There are a few things which are of value in differentiating some of these types of jaundice. For instance, we all know that pain is one of the characteristics of obstructive jaundice, particularly when jaundice is due to a calculus in the common duct. However, the corollary does not necessarily follow that in non-obstructive jaundice pain is absent; nor does it follow that in all cases of obstructive jaundice is pain a predominant symptom. We not infrequently have observed patients who have had common duct stones and a true obstructive jaundice who have had no more pain than those patients in whom calculus was not present and the obstruction was due to a malignancy of the head of the pancreas or the ampulla of Vater or some other lesion usually characterized by so-called painless jaundice.

What are some of the important factors which one may consider in the differential diagnosis of jaundice? I do not know what all of them may be, but I think that a very important matter is a careful history and physical examination. Oftentimes one obtains excellent leads regarding the differential diagnosis of jaundice just by a careful history and physical examination. In the history, the important factor, of course, is the presence or absence of pain. Also, a history of a familial type of icterus, the chronicity of the icterus, enlargement of the spleen and fragility of the red cells usually justify the diagnosis of hemolytic icterus. In some patients with jaundice the size of the liver in relation to the degree of jaundice and the duration of the jaundice are enlightening in relation to the differential diagnosis of the various types of jaundice. To ascertain or determine that there is a mass in the upper right quadrant of the abdomen which may be interpreted as a distended gallbladder is of importance in the differential diagnosis of some of these cases of obstructive jaundice. I think we are all agreed that in the absence of pathology in the gallbladder, in the absence of calculus disease, and when the gallbladder has not previously been removed, Courvoisier's law usually applies in malignant obstruction of terminal portion of the common duct by virtue of a lesion of the ampulla of Vater or as a result of a malignant lesion of the head of the pancreas.

Those, in brief and very rapidly and quite superficially, we might say are the clinical manifestations obtained in the history and in the physical examination which are important in determining or at least arriving at some sort of opinion in the differential diagnosis of the various forms of jaundice.

Certain laboratory aids are of value in differentiating obstructive and non-obstructive jaundice. It is possible to determine whether or not any patency of the common duct exists and whether or not bile is passing into the intestinal tract from the common duct. There are a number of ways in which this may be ascertained. One of these is a test for urobilin in the stool, and another is the determination of presence or absence of bile pigment in the duodenal content. We all know that in the absence of bile pigment in the duodenal content, the duodenal content is a clear mucoid substance entirely devoid of any coloring matter or pigment whatsoever.

In the evaluation of the finding of the absence or presence of urobilin in the stool or bile pigment in the duodenal content error may occur in the interpretation of these findings. One should bear in mind that there are many cases of so-called hepatogenous type of jaundice in which by virtue of the chronicity of the disease and the changes which have occurred in the parenchymatous tissue in the liver, the excretory function of the liver has been abolished in relation to bile pigment. At any rate one encounters a total absence of bile pigment in the duodenum in certain instances of prolonged intrahepatic type of jaundice. Therefore, it must follow that one should not necessarily make a diagnosis of obstructive jaundice simply because absence of bile pigment in the duodenal content has been demonstrated.

There are lesions which produce complete obstruction of the common duct. However, we should bear in mind that not all obstructive lesions of the common duct produce complete obstruction. We all have seen instances in which some patency of the common duct persists. As an actual matter of fact, some patency of bile ducts persists in practically all cases of calculus obstruction of the common duct, so that some bile is seeping down through past stones into the common duct, and bile pigment can be recovered in the duodenal content under those circumstances. By the same token urobilin will be obtained in the examination for it in the stool. All of these various examinations are relative in their importance so far as the differential diagnosis is concerned and they must be evaluated in relation to other clinical and laboratory data in order to avoid the greatest percentage of error.

You all know about the van den Bergh, direct and indirect, and I am perfectly willing to state that you know more about it than I do. As an actual matter of fact, I have found these tests of no value whatsoever in differentiating the non-obstructive from the obstructive type of jaundice. I realize that there are many in this audience who will not accept that statement nor agree with me. I am perfectly willing to state that so far as my own experience is concerned, the icteric index and the bilirubin content of the blood are of service to me only in determining the degree of the jaundice and, upon repetition of those tests, the progress of the jaundice.

There is a very interesting exhibit down in the scientific section on this floor by Steigmann, Popper and Meyer. Those of you who are interested I am sure will derive much of value from reviewing that exhibit on the various laboratory data which have been employed in a large series of cases at the Cook County Hospital in an effort to differentiate the various types of jaundice.

Occasionally after one has studied these patients very carefully and has employed all the laboratory aid that is available to one, it still is impossible to make an accurate differential diagnosis between an obstructive and a non-obstructive type of jaundice. There are certain instances in which the weight of evidence is in favor of a non-obstructive or a hepatogenous type of jaundice. Still, one is unable to exclude definitely the question of an obstructing lesion. Under these circumstances the question arises as to what to do about it.

Bloomfield has made observations on quite a series of borderline cases in whom there was a good bit of question regarding the diagnosis, and he has arrived at the solution of the problem through observation of these patients for fully six weeks, particularly if there is any trend toward diminution in the degree of jaundice. I am not wholly in agreement with such a program. At the same time, in many instances there is no grave objection to it. I think, however, that in most instances it is possible, particularly where the probability of an obstructing lesion may be justifiably entertained, that one can get around to the idea of making up one's mind about it under six weeks. In the majority of instances one should, because there are certain effects of prolonged jaundice which one wishes to avoid if it is possible to do so. Early surgical exploration is highly in order when the weight of evidence favors an obstructive type of jaundice. In all questionable cases in which an obstructing lesion cannot be excluded surgical exploration should at least have due consideration.

What are the effects of prolonged obstructive jaundice or of prolonged jaundice? We all know that in jaundice the metabolism of fats, proteins and carbohydrates is materially altered. It is an interesting observation that the greater the degree of jaundice, particularly in the obstructing lesions and the greater the degree of obstruction, the more rapidly do these alterations in metabolism of fats, proteins and carbohydrates occur. That is particularly true in the obstructing lesions of the terminal portion of the common duct where there is not only obstruction of the common duct and obstruction to the outflow of bile, but also obstruction to the external pancreatic secretion. We all have seen patients with obstructing lesions of the terminal portion of the common duct, particularly malignant lesions of the ampulla of Vater or the head of the pancreas, decline very rapidly. As a matter of fact, loss of pancreatic secretion causes their decline much more rapidly than simply the loss of bile excretion.

Ravdin was one of the first, or at least one who with impressiveness first brought to the attention of the profession the effect of jaundice upon the liver, particularly as pertains to the disturbance of carbohydrate metabolism and the disturbance of the metabolism of proteins in the presence of obstructive jaundice. He has recommended, as have others by this time, and it has become universally the practice in the preoperative management of patients with obstructive jaundice to employ a diet high in proteins and high in carbohydrates to the exclusion of fats. We all know that in the presence of prolonged obstructive jaundice there is a marked increase in the blood lipids and an increase in the liver lipids, and I think that in many instances in the past in which we have had so-called lethal results or death following cholecystectomy which has been unexplained, death has been due to hepatic insufficiency as the result of fatty infiltration or fatty degeneration, whichever way you wish to speak of it. Hypoproteinemia has only been emphasized in recent years as an important factor in relation to the safety of surgical procedures in obstructive jaundice.

We may say, in general, that after careful study any patient in whom one may legitimately entertain a diagnosis of obstruction of the bile ducts, surgical exploration is worthy of consideration. I should furthermore say that in view of the relative safety with which operation may be performed upon the various obstructing lesions of the common duct, it is incumbent upon us to develop a policy of early surgical exploration in any case in which doubt exists regarding the nature of the lesion, or doubt exists as to whether an obstructing lesion of the common duct is present.

There are those who because a patient does not give a typical history of biliary colic immediately arrive at a diagnosis of a neoplastic lesion of the duct and immediately deny the patient the privilege of surgical exploration on the basis, first, of assumed accuracy of the diagnosis, and secondly, assumed futility of any surgical procedure. Dr. Cattell yesterday showed conclusively that some of these lesions in the terminal portion of the common duct can be operated upon with a fair degree of safety to the patient and with a fair degree of assurance of not only palliation but cure of the disease.

We all know that calculous disease of the common duct is the most frequent form of common duct obstruction. There is a great deal of discrepancy in the various reports regarding the incidence of common duct stones. One of the most convincing observations which has been brought to the profession during the past ten years has been that contained in the report of Lahey and his associates from the Lahey Clinic in which upon careful exploration of a large series of patients with calculous disease of the gallbladder, there was an incidence of common duct stone in approximately 20 per cent of the cases. Allen found, during a four-year period at the Massachusetts General Hospital, an incidence of common duct stone in 20.7 per cent of the cases of calculous disease of the gallbladder. In our own work at St. Vincent's Hospital in Los Angeles during the past four years we have opened and explored the common duct in 44 per cent of the cases of calculous disease of the gallbladder and have recovered one or more stones from the common duct in 19 per cent of the cases or in nearly half of the patients in whom surgical exploration of the common duct was employed, which brings up the question: What are the indications for exploration of the common duct? First of all the common duct should be explored in every patient who is jaundiced at the time of operation. All patients who have had a history of jaundice incident to biliary colic at any time previous to operation should have the interior of the common duct explored. Also, all patients in whom small stones one or two millimeters in diameter exist in the gallbladder, whether or not any are impacted in the cystic duct, particularly if the cystic duct is dilated, should have exploration of the common duct. Furthermore, it goes without saying that any patient in whom a stone is palpable in the common duct it should be removed. Another most important indication for exploring the common duct is undue dilatation of the common duct; a common duct that is larger than normal. It is interesting that in the absence of any other clinical manifestations, undue dilatation or enlargement of the common duct oftentimes is due to one or more stones in the common duct.

It is the general opinion among many men that jaundice is always manifested in the presence of one or more common duct stones. It is worthy of note that in 26 per cent of the patients in whom we have removed one or more common duct stones the patient was not jaundiced at the time nor was there a history of jaundice at any time previously. So one cannot always rely upon the history of jaundice. One should bear in mind that there is a certain percentage of patients who actually harbor stones in the common duct who are not jaundiced at the time nor have they been jaundiced at any time previously.

An important matter in the mind of everyone is the question of the value of cholangiograms when dealing with common duct stones. There are those who are enthusiastic about making cholangiograms at the operating table to be certain that all stones have been removed and that the ampulla of Vater is patent. Personally I do not believe that this is necessary except in a small percentage of instances. It is usually possible to pass scoops and probes up into both hepatic ducts and down through the ampulla and thereby provide reasonable assurance that all stones have been removed from the hepatic and common ducts. There are certain instances, however, in which postoperative cholangiograms are advisable. So far as we are concerned, we make them only in cases in which we are performing secondary operations or in patients in whom there may be some doubt, and usually those are patients in whom we institute T-tube drainage of the common duct.

I wish to discuss very briefly two other lesions of the common duct. One of these is postoperative stricture of the common duct. The atresias and the strictures of the common duct which surgeons see are the result, for the most part, of traumatic injury to the common duct incident to cholecystectomy. There is only one way to avoid such atresias and strictures and that is through an intimate knowledge of the anatomy and of the anomalies of the ductile and vascular structures of the biliary tract and their accurate visualization in performing cholecystectomy. It is much easier to avoid injuries than it is to repair them. Many procedures have been instituted in the repair of these injuries. In my experience over a period of years the most satisfactory type of operation which has produced the best results has been an anastomosis of the common duct above the stricture at the hilus of the liver or at some level just above the stricture to the duodenum as an hepaticoduodenostomy.

(Slide) We shall come back just a moment to the value of cholangiograms. These are normal cholangiograms which we have made postoperatively in patients in whom we have performed secondary operations. We make cholangiograms primarily for the purpose of record, to have a record that the common duct and the ampulla are patent, and you will see that these are normal cholangiograms for the most part.

(Slide) Here is another one in a secondary case. A patient had been operated upon three times previously and it was of great value to us to have a record subsequently that we did have a patent common duct.

(Slide) Here is an abnormal cholangiogram. It really is not a cholangiogram. It is lipiodol injection of the common duct in a patient we saw as a result of a biliary fistula, who had been operated upon previously for this fistula which had been draining for months. This is simply the injection of lipiodol into the sinus, which fortunately outlined the common duct which harbored a large stone.

(Slide) Here is another patient who had a biliary fistula. The injection of lipiodol into the gallbladder filled out the lower part of the common duct and indicated an obstructed ampulla. The obstruction proved to be due to a benign tumor of the ampulla of Vater.

(Slide) Here is an abnormal cholangiogram. I had performed a cholecystectomy upon this patient in the presence of jaundice and had determined upon exploration of the common duct, to my own satisfaction, that this patient had a pancreatitis. However, because of the fact that bile did not appear postoperatively in the duodenal content and in the stool, we had this cholangiogram made, and the finding of obstruction at the terminal portion of the common duct in conjunction with blood in the stool allowed us finally to arrive at a diagnosis of a neoplastic lesion of the ampulla.

To proceed hurriedly to the question of stricture, I present these slides simply to show the methods of repair by hepaticoduodenostomy over a tube, making a cuff distally extending into the duodenum and a cuff extending up into the common duct with the anastomosis between the common duct and the duodenum. The cuffs are for the purpose of preventing the tube from slipping back into the common duct or down into the duodenum too early.

(Slide) Here is another slide showing the various size tubes. These tubes are made of ordinary catheters and the size of the tube in the common duct is dependent upon the size of the common duct.

(Slide) The question arises as to how long these tubes should remain in place. I cannot answer the question except to say that here is the slide of a patient upon whom I operated twelve years ago. This patient had a biliary colic five months and ten days after the operation extruding the tube at that time, and the film here shows the tube passing out into the jejunum.

(Slide) This is a film of the same patient two and a half years afterwards, showing complete patency of the ducts with air from the duodenum in the biliary tract.

(Slide) Some of these tubes have passed a little too early and before they had accomplished their purpose. In one patient the tube was extruded on the eighth day as a result of biliary colic, and the patient subsequently developed a stricture and had to be operated upon again. During the past year in several cases we have passed a silk suture through the tube in the common duct and have anchored the suture to a pearl button on the abdominal wall for as long as six months, thereby providing assurance that the tube in the common duct will not be extruded until the anchoring suture has been released.

A few slides will illustrate the surgical procedures employed in neoplastic obstruction of the terminal portion of the common duct. Many of you have seen these slides as they have been published elsewhere. There are certain lesions of the terminal portion of the common duct which can be removed by the transduodenal route. This is one of those, and this slide shows the method of transduodenal cautery excision of the lesion which can be done in the small non-infiltrating lesions. The severed pancreatic duct and the common duct are shown here. These were subsequently reimplanted into the posterior wall of the duodenum.

I might say that we have operated upon four patients in whom transduodenal removal of a lesion at the terminal portion of the common duct was accomplished. Two of them were malignant and two were benign lesions. This slide shows a tube which was used in the common duct and at the site of the reimplantation of the common duct to avoid stricture. We have not had stricture of the common duct at the site of reimplantation occur in these patients. However, several years ago when I reviewed 124 cases of carcinoma of the ampulla of Vater in which a certain percentage of cases had been operated upon by transduodenal excision it was found that a postoperative stricture did develop at the site of the implantation of the common duct into the posterior wall of the duodenum in a number of cases.

(Slide) There are other more extensive infiltrating lesions of the terminal portion of the common duct such as this. We operated upon this patient in March, 1940, and this slide shows a huge lesion which upon exposure proved to be too large and too extensive to remove by transduodenal excision, so a resection of the duodenum and the head of the pancreas was employed. I thought at that time, March, 1940, that this was the first time the Whipple two-stage operation had been performed in one stage. My operation was performed in the third week of March, 1940. However, Allen Whipple performed his two-stage operation in one stage two weeks previous to that. This slide simply shows the completed operation of cholecystogastrostomy, closure of the stomach, the pancreas and the jejunum and a posterior gastro-enterostomy. This patient is living and perfectly well three and a half years after operation.

(Slide) A modified one-stage Whipple radical procedure was employed in the second case. The slide depicts the lesion in the inset. Resection of the head of the pancreas and the entire duodenum was instituted and instead of closing the severed pancreas and thereby sacrificing the external pancreatic secretion, the severed pancreas was capped by the open end of the jejunum as a pancreatojejunostomy. Closure of the pyloric end of the stomach, transplantation of the common duct into the jejunum, and posterior gastro-enterostomy completed the operation.

(Slide) Sagittal section of the specimen shows the head of the pancreas and retroperitoneal lymph nodes involved in the malignancy, which were removed with the lesion en bloc. This patient is living and well two and one-half years after operation.

(Slide) This slide shows the lesion of a third patient with a periampullary carcinoma producing obstructive jaundice, operated upon two years ago this month. The mucosal side of the duodenum and the sagittal section of the pancreas are presented in this slide. The lesion is a true ampullary carcinoma.

In conclusion it may be stated that the surgical management of these patients has advanced to such a degree through the use of vitamin K and through the preoperative management restoring the liver glycogen and improving the hepatic function that these patients may be operated upon with a greater degree of safety than at any time previously, and I think we are justified in adopting the policy of operating upon all cases of obstructive jaundice.

DIAGNOSTIC UROLOGICAL CLINIC

Urologic Lesions Causing Abdominal Symptoms in Children

By MEREDITH F. CAMPBELL, M. D.
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Because of the limitations of time we will confine our remarks to pain as a symptomatic manifestation in children and as it may tend to confuse abdominal diagnosis.

(Slide) The chief causes of abdominal pain of urinary tract origin in children are the obstructive lesions which are predominantly congenital and they may range from a congenital stenosis of the external meatus to a stricture of the renal calix.

Here are listed the common urologic conditions in children, many of which are congenital or the result of congenital obstruction. Of these, the obstructive lesions of the ureter are by far the most important, particularly when the lesion is on the right side, as the symptoms they produce are apt to confuse the diagnosis, causing the erroneous diagnosis of appendicitis to be made.

We appreciate that in an infant or child with symptoms of acute intra-abdominal disease, laparotomy is usually indicated, even though the precise diagnosis may be in doubt. We are particularly interested here with the cases of persistent or recurrent intra-abdominal pain which are not emergency and in which we urge that a urologic examination be carried out at least to the extent of doing an excretory urographic study. We consider that a physical examination and urinalysis (using only a catheterized specimen from the female), and an excretory urographic study, represent the minimum of urologic investigation.

In studying these cases, the intravenous pyelogram will sometimes enable one to make an accurate anatomic diagnosis. Where there is any question as to the anatomic diagnosis as concerns the urinary tract, further study should be made employing cystoscopy, ureteral catheterization, and retrograde pyelography. The illustrative cases we are going to show you simply point up this dictum; all of these lesions are more or less common, but I would direct your special attention to congenital obstruction of the upper urinary tract, chiefly stricture.

Here we have a four-year-old boy with pain in his right side, persistent pain, as a result of a congenital hypoplastic fibrotic kidney. Several of these cases have been reported in adults, not so many in children. The pain is very apt to be referred to the mid-abdominal region and cause an erroneous diagnosis of intra-abdominal disease to be made. Nephrectomy is usually necessary.

(Slide) This patient's lesion is much more common, vascular obstruction of the ureter, particularly near the uretero-pelvic junction. Occasionally the vascular obstruction is at the lower end of the ureter where it is usually an anomalous uterine vessel.

The child, five years old, was admitted to the hospital with persistent pyuria but, more important, persistent pain in the right lower abdominal quadrant. She had had it for four years. When she was two years old, the appendix had been removed, presumably because of so-called chronic appendicitis. Yet when she was admitted to the hospital at the age of five, we found her kidney was entirely destroyed by obstruction and complicating infection; a congenital aberrent renal vessel passed from the lower renal pole to the aorta, and in the pyelogram the filling defect caused by the vascular compression shows beautifully. It was necessary to remove the kidney.

We have seen some twenty of these children and eleven of them were right sided. In seven the appendix had been removed because of the pain for which the child subsequently again came to the hospital.

This next child, a girl eight years old, had persistent pain in the gallbladder region for many months. Her pediatrician was sure she had gallbladder disease. She was subjected to eight complete cholecystographic studies and three complete gastrointestinal x-ray studies. Finally it was suggested that an excretory urographic study be made, which showed a large hydronephrosis on the right side. This examination was followed by retrograde pyelography which demonstrated the horseshoe kidney disease from which she suffered with massive hydronephrosis of the right segment. Treatment was simply resection of the horseshoe kidney, leaving the good left renal segment. Immediately thereafter, all of her symptoms disappeared. Subsequently the urine was sterilized. The child gained weight and had a very satisfactory result.

(Slide) This shows the surgical anatomy in the foregoing case. One of the interesting findings was the large renal vein which came down and bifurcated to supply each side of the isthmus.

(Slide) The next patient illustrates another form of horseshoe kidney disease. This nine-year-old boy was admitted to the hospital with a temperature of 105° F., acute pain in the right lower quadrant and the clinical picture of acute appendicitis. He was explored; the surgeon at once encountered a large pool of purulent urine, inserted a drain, and backed out. The boy continued to drain urine for several weeks and eventually came to our hands, and on urological examination we found a pelvic horseshoe kidney with the left half overlying the sacrum. The right half passed up into the right lumbar gutter and showed massive hydronephrosis due to congenital stricture of the pelvic outlet. There was severe complicating renal infection. The mechanism which precipitated the acute attack simulating appendicitis was acute obstruction at the ureteropelvic junction with exacerbation of the infection and acute hydronephrosis. Treatment was resection of the kidney, leaving the good left half.

(Slide) The next patient was a twelve-year-old girl admitted to the hospital because of pain in the lower abdomen and also a mass. When she lay on the table you could see a suprapubic protrusion as though she had a full bladder. This was found to be a huge pelvic fused kidney overlying the sacrum. The schema shows the local anatomy. The pain was due to the continuous drag on the anomalous renal vascular supply and this pain was relieved by an abdominal support which elevated the kidney and relieved the vascular tension.

(Slide) Here is another type of renal fusion in which the left kidney is fused with the right kidney over in the right lumbar gutter. A month before we saw this child she had an appendectomy under the diagnosis of acute appendicitis. A normal appendix was removed. Subsequently she had a urologic examination which disclosed a stricture at the right ureterovesical junction. By instrumental dilatation of this stricture her symptoms have been permanently relieved.

(Slide) A female child six years old was admitted to the hospital because of recurrent pain in the right loin simulating intra-abdominal disease. It was found on urologic examination that she had a malrotation of the right kidney causing periodic obstruction at the ureteropelvic junction, hence the recurrent abdominal pain.

(Slide) This twelve-day-old boy was admitted to the hospital with the clinical picture of acute appendicitis; he was exceedingly ill. The only thing that caused delayed laparotomy was the fact that he had a little blood in the urine; he had just a little blood in the stools. This retrograde pyelogram was made when he was twelve days old and you see the kidney pelvis was completely blocked out. Next day the kidney was explored and it was found that he had a massive infarction of the kidney. Nephrectomy was performed. His clinical picture was rather typical, that is, ileocolitis preceding the acute attack of pain in the loin and due to the secondary renal infarction. Within six weeks we encountered the identical picture in a boy in whom we performed nephrectomy at thirty-three days of age. Both made splendid recoveries. Certainly in each instance, and without urologic investigation, the clinical picture would normally lead one to explore the abdomen.

(Slide) An eight-year-old girl had a so-called silent stone in her right kidney pelvis, silent except for recurrent attacks of mild pain which were usually localized in the gallbladder region. It was a coral stone completely filling the kidney pelvis and it was necessary to remove the kidney.

(Slide) The next lesion, stricture of the ureter, is much more common, and is of special clinical interest when it involves the lower half of the ureter or in the region of the junction of the middle third and the lower third. The diagnosis of chronic appendicitis will probably be made in four out of five cases, as it was in the following case. This child was going to be subjected to appendectomy, but an excretory urographic study was made and the right ureteral stricture was detected. Her "chronic appendicitis" was cured simply by repeated periodic dilatations of this stricture with bougies. She has been free of symptoms now for over five years.

(Slide) This five-year-old girl with so-called chronic appendicitis, had her appendix removed but her pains persisted and were finally cured by dilatation of the stricture at the junction of the lower and middle thirds of the ureter. In the ureterogram you see the dilatation above the point of obstruction, which anatomically is essentially behind the appendix.

(Slide) The same clinical picture is here demonstrated in a two-year-old child with repeated attacks of lower right quadrant pain thought to be due to acute appendicitis. On urological examination congenital stricture at the junction of the middle and lower thirds of the ureter was readily demonstrated. The lesion was easily controlled by ureteral dilatation and the symptoms eradicated.

(Slide) In this next patient we have the same thing on the left side in a five-year-old child who had recurrent attacks of left loin pain. It was thought she had surgical disease of the descending colon. The excretory urographic study showed this pyelogram

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which demonstrated a tight stricture in the body of the left ureter. Subsequently a retrograde pyelogram was made which was confirmatory. Treatment: ureteral dilatation.

(Slide) Here is another child six years old with the same condition on the right side, admitted to the hospital with a diagnosis of subacute appendicitis but was successfully relieved by ureteral dilatation.

(Slide) A seven-year-old girl was admitted to the hospital with intermittent pain in the left upper abdominal quadrant. She was found to have a mild hydronephrosis above a stricture of the ureter at the ureteropelvic junction and at the ureterovesical junction. The only therapy that was permitted by the patient was dilatation of the lower end of the ureter.

(Slide) The massive hydronephrotic kidney shown on the screen was removed from a four-year-old boy who had a congenital left ureteropelvic junction stricture, persistent pain in the left loin, later a palpable mass, chiefly anterior in the left upper quadrant.

(Slide) A more unusual lesion was found in a five-year-old girl with persistent pain in the left loin, especially toward the left lower quadrant. It was thought that she might have a left sided appendicitis, yet urological examination revealed a diverticulum of the left lower ureter. As the diverticulum became distended with urine, it compressed the ureter, caused ureteral and pelvic back pressure which in turn caused the pain of which the patient complained. The diverticulum was excised and symptoms were promptly relieved.

(Slide) The next case illustrates another type of anomaly, abnormally high insertion of the ureter. This lesion was on the left side in a five-year-old girl with continuous pain in the left upper quadrant and thought to have intra-abdominal disease. There was no treatment permitted here; plastic reinsertion of the ureter has been employed in some cases.

(Slide) Urinary calculus is not particularly common in children in this country and doubtless because of improved dietetics. The four-year-old boy here demonstrated was admitted to the surgical service of the hospital. Everyone who examined him said they would say he had a ureteral calculus, except "you don't have that in children so young." It happened in this boy, and he had this triangular calculus originally in the upper ureter. We dilated the ureter conservatively, hoping he would be able to pass the stone. The calculus finally got down to the region of the seminal vesicle, and since it progressed no farther in three weeks, open ureterotomy was performed and the boy was relieved. When ureteral calculus occurs on the right side in children, the diagnosis of acute appendicitis is almost always made, and unfortunately too often so when the patient is an adult.

We saw a patient illustrating this diagnostic difficulty not long ago, a ten-year-old boy. Ten days previous he had a gangrenous appendix removed. Ten days postoperatively he was taken suddenly with severe abdominal pain, cramps, vomiting, and signs of intestinal obstruction on the left side. During one of these attacks he had a little pain in his bladder and a little frequency of urination. Intravenous urographic study was suggested. This showed a stone in the left lower ureter about the size of a grain of rice, which he fortuitously passed about two hours after the excretory examination and with complete relief of his pain. I mention this patient to show how the judicious employment of such a simple method as excretory urography may save the child exploratory laparotomy at a critical moment.

(Slide) Finally, and getting down to the lower urinary tract, we were surprised to find in going over a series of 152 children with congenital stenosis of the external urethral meatus, a congenital stricture of the external meatus, if you will, that in nine severe abdominal pain was the chief complaint. They had urinary frequency and some difficulty of urination, but lower abdominal pain was the chief complaint. Here is a cystogram in an eighteen-month-old boy who had that condition, and you see the enormous dilatation of the bladder, which, of course, caused the pain. The back-pressure damage had progressed to cause rather marked dilatation of the entire upper urinary tract. In such a case the treatment is simple enough—liberal meatotomy and the maintenance of a wide-open urethral canal.

One fact I would especially like to leave with you is that while the urinalysis in serious urinary tract disease will usually be abnormal, occasionally you will see severe urologic disease and most often advanced obstructive uropathy, with a normal urinalysis.

It would seem that the slides that have been shown you and the cases that have been illustrated are sufficient to indicate the advisability of taking out twenty to twenty-five minutes' time for an excretory urographic study in patients, not only children but adults as well, in whom the diagnosis of chronic appendicitis seems a likely one. This procedure is employed in most teaching hospitals in this country today. In short, no patient should be operated on under the diagnosis of chronic appendicitis or abdominal adhesions until disease of the urinary tract has been ruled out.

DIAGNOSTIC SURGICAL CLINIC

BILIARY SURGERY

By JOHN F. ERDMANN, M. D.
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I am delighted to greet you again, gentlemen.

We have three cases that we will show and read the histories of each of them and then make comments after the patients have been discharged from the room.

The first patient is a female thirty-two years of age. She entered the Illinois Research and Educational Hospital on September 9, 1943, complaining of yellow color to her skin, itching, attacks of right upper quadrant pain radiating to the right shoulder, occasional acholic stools, dark urine, and a weight loss of eighty-two pounds up to May, 1943. This patient had been having rather typical gallbladder attacks since 1937. These were precipitated by eating fried foods and usually lasted from one to five days, associated with nausea and vomiting. The attacks were relieved by morphine. A rather typical and characteristic onset or history of gallbladder disease.

On the fifth day of last January she was brought into the hospital in Peoria, Illinois, because of a severe attack of pain. On the 26th day of that month she was operated on and was told that a gangrenous gallbladder was removed, and some stones were removed from the common duct. A T-tube was introduced for drainage. Her postoperative course was evidently uneventful and she felt good after the operation and gained twenty pounds, until the following April when she extracted the T-tube by accident and from then on her trouble began. She started to have itching of the skin and in May noticed she was getting quite yellow and began having attacks again in the right upper quadrant. She noticed that her stools were light in color.

Physical examination of the patient was essentially negative except for the icterus. Routine laboratory work was also essentially negative.

During her preoperative hospital stage she had several attacks of acute colicky right upper quadrant pain radiating around to the back, associated with nausea and vomiting and slight tenderness.

On the first day of October she was operated on by Dr. Cole, who performed a right paramedian incision. There were numerous adhesions in the right upper quadrant. These were separated, exposing the region of the common duct. A bulging stump of proximal common duct measuring 2 or 3 cm. long was found. It is usual in my work to find a bulging stump of the proximal end. A stricture was found at the terminal end of this stump. Further dissection revealed the terminal end of the common duct with a defect of about 1 or 1.5 cm. intervening. A probe was placed in the distal end. This was followed by a total failure. The probe did not go into the duodenum, so he incised the duodenal wall over the probe and dilated the fibrotic area. He then took the end of a catheter and threaded it up into the distal end of the common duct, allowing about three inches to remain in the lumen of the duodenum. He then closed the opening he had made in the duodenum and placed a vitallium tube between the two ends of the common duct at the constriction. I wish I had a vitallium tube here, but I have been down to the instrument display on two different occasions since I have been here, and one did not know what a vitallium tube was and the other did not have one in the house although they were the authors of it.

The wound was then closed over with adjacent tissue.

The second case was a white female twenty-three years old when she was admitted to the hospital, complaining of attacks of a grinding, dull pain in the epigastrium which in about ten minutes would change to a sharp pain and radiate around the right costal region and go straight through to her spine. Also a characteristic case of stones in the gallbladder. At the time of admission to the hospital the attacks were a daily occurrence. These attacks were not relieved by nitroglycerin but by morphine. Evidently he was searching for one of the dyskinesias we see every now and then relieved by an antispasmodic such as nitroglycerin and some of the nitrates. Nobody wants to operate on a dyskinesia case if he can avoid it, on account of the after results, of course.

On admission her routine chemistries were normal and serum amylase was 50 mg. per 100 cc. On the 7th of September the patient had a severe attack and spiked a temperature of 104. Subsequently on the 20th of the month the abdomen was opened through a right rectus incision and a large indurated pancreas was found and a gallbladder with stones removed. Her postoperative course was uneventful. She had, in other words, associated with her cholecystitis a definite pancreatitis.

Subsequent clinical followup showed her wound to be healed solid and the epigastric pain to be entirely relieved.

Operation by Dr. Cole: right paramedian incision. The gallbladder was surrounded by a few adhesions in the lower portion and contained a few stones. The pancreas was indurated and enlarged about two times. It was obviously the seat of a subsiding, acute, edematous pancreatitis. The gallbladder was removed from below upward. The common duct was not enlarged, and was therefore not opened, particularly since no stones were palpable within it. The wound was closed around a drain and she has made a perfect recovery and has gained in weight.

This gallbladder was removed from below upward. I don't care whether I remove the gallbladder from below upward or above downward or a combination of both; it is just a question as to what happens to strike me as the most feasible thing to do at the time, but in the majority of instances I excise the gallbladder from below upwards after I have located the cystic duct without any question in my own mind, and it is a simple thing to do.

The third patient is the man on the left who is forty-eight years of age. He was operated in March 1942, one year and six months ago and he has now bile draining from his abdominal wound. This patient states that he was well until January 1942 when he noted he was losing his appetite, his skin was becoming yellow, his stools were becoming lighter and his urine was bronzing in color, itching, and no pain. The first thing you would think of here is what Dr. Hunt spoke to you about this morning, the possibility of a malignancy: no pain onset, gradually increasing icterus, itching of the skin, and subsequent to that skin marks of the contusions which readily show up, and hemorrhagic spots. Nevertheless, he was operated upon in March of 1942 at another hospital. Report: cholecystogastrostomy for a tumor at the head of the pancreas. The patient was well until December 1942, when symptoms reported above returned, plus a series of chills and fever, followed by dull ache and "flu"-like malaise. Remember, he had a cholecystogastrostomy. Following the cholecystogastrostomy he had chills and fever and malaise. The question now comes up: Did this man have at that time an ascending cholangitis, which is found quite frequently after cholecystogastrostomy, or did he not? I would rather be inclined to think that he had an ascending cholangitis from his cholecystogastrostomy. Nevertheless, there are times when we must do a cholecystogastrostomy.

He was operated on again in May of 1943 at another outside hospital and the tumor still appeared benign. The common duct was much dilated. A T-tube was placed in the common duct and the tube was pulled out accidentally six weeks ago and bile has been draining on pads ever since, which require changing quite frequently. Since this operation there has been no return of any of the original symptoms.

He was admitted to the Illinois Research Hospital on the 24th of this month and stated that he had typhoid thirty years ago. A great deal of importance was attached to the statement that the patient had typhoid in early life. Typhoid was considered some twenty-five years ago by most of us surgeons and also diagnosticians as being a primary cause of the production of stones, finding the bacillus of typhoid in the center of these concretions. This argument or theory has been washed out pretty thoroughly.

He had acute rheumatic fever at the age of fourteen. He had a skull fracture forty-one years ago. One brother died at the age of forty-seven of diabetes.

His loss of weight is seventy-five pounds since January 1942. The stools since May 1943 have been brown, never tarry, but intermittently diarrheal.

He is in the hospital at the present time with an operation staring him in the face.

Let us take these up in the order of the first shall be last and the last shall be first. The man who has just gone out evidently has a leak from his common duct. What the obstruction is time and exploration alone will tell, whether he has a malignancy there or not. It didn't sound to me in the beginning as though this man had a malignancy of his pancreas or at the ampulla of Vater, very beautifully shown and demonstrated to you by Dr. Verne Hunt this morning.

He evidently is being prepared for secondary work.

Mrs. Margaret Grausso is brought in purely as a case to show you the combination of cholecystitis, either chronic or acute, with an added acute pancreatitis. You know that in cases of cholecystitis with pancreatitis of the acute variety, it is very difficult to isolate the common duct in many of these patients on account of the edema and the infiltration around about. It is just this type of case in which we have the subsequent or secondary manifestations of stone left in somewhere or other. So far this girl presents none of those manifestations whatever.

The first woman pulled out her T-tube and began to leak and got her second dose of operative work and apparently is well at the present time.

So much for the clinical side.

Surgery of the gallbladder involves the acute cases and the chronic cases, so-called, and then the dyskinesias. In the dyskinesias if you cannot liberate your pain by means of one of the antispasmodics or one of the nitrates or nitrites and the x-ray is negative, there are times when it pays to go in and explore. If it is dyskinesia pure and simple, one of the spasms of the sphincter, the chances are about forty per cent it will not be improved, differing a little bit from the so-called strawberry gallbladder which is removed where you have about eighty per cent improvements with about twenty non-improved.

The acute gallbladder is a controversial point in the medical press and in all medical societies. What are you going to do when you get one? Are you going to ice-pack it for several days until the temperature subsides and give the patient a chance to run away and have an operation at some later time, or are you going to approach that patient and state to him, "You need an operation at the present time and will withstand it just as well, in my opinion far better, now." When you have an inflammatory condition to operate, are you going to wait until you have an absolute subsidence and loss of all the inflammatory manifestations? Robert T. Morris used to take the ground that when you had an inflammatory condition your army of defense was well organized and in order; when you did not have an inflammatory condition you might produce an army of offense, and I still take that ground. In other words, when I see an acute gallbladder characterized by the temperature, the pulse, the blood count, tumor present or absent, exquisite tenderness in the upper right quadrant, I put that person in the hospital just as soon as I can provided he will consent, and the majority of such patients will consent.

In the chronic or so-called chronic gallbladder, one with recurring manifestations, we have a different thing to contend with. The argument there is that if stones are present the stones may produce malignancy. God forbid that I should ever use that term in my office; nevertheless we do know by our studies that malignancy is inducted in the majority of instances where stones are present. Let me reverse it and say that stones are productive of malignancy in about ninety-eight to ninety-nine per cent, or, to explain it differently, in every malignancy that we have found in my personal service there have been stones or sand present in the gallbladder.

The late Dr. Deaver reported about ninety-six per cent of all patients with stones as subsequently being involved by malignancy, and he used the argument very strongly that the presence of stones was a factor in the production of malignancy.

When you get a chronic patient, that is, one who has the so-called chronic gallbladder, stones, belching, bloating, recurrence of attacks, loss of weight, which I have had in my own service as high as forty-five pounds without any inflammatory condition whatever (one of the diseases with the largest loss of weight is cholecystitis with cholelithiasis), the question always arises as to operation. Do you advise operation with the argument that there is a malignant tendency here or a malignant possibility, or do you withhold your argument and say, "Wait until you get an acute attack," or do you say, "Do you suffer very much? If you do and you dread these attacks and have to take large doses of morphine, I would advise early operation." That is my argument with all of them. I do not use the argument of malignancy being imposed upon stones. I don't like the word any more than I like the word "adhesions." But I do say to them, "You are losing weight. If you are having one attack of pain after another, you are a terrible case of unrest to yourself and more or less to your husband or wife and to the rest of your family. Why don't you take the matter in hand and be operated upon and get through with it?"

The question of jaundice was so very beautifully taken care of this morning that all I wish to do is to emphasize that jaundice which is slow in its onset, not remitting, constantly rising or holding at one point, no pain, loss in weight, a sense of enfeeblement, should cause you to look out for malignancy. It is slow, non-pain-producing jaundice that I fear always from the standpoint of malignancy.

Of course, there are jaundices which are remitting, and when you have an intermittent jaundice, a rise and fall in the intensity of it, or a period of jaundice and a period of absolute lack of jaundice for some length of time, you simply have a stone or some allied condition of partial duct obstruction—operation is not demanded so much as it would be in malignancy. Nevertheless, that stone is not going to pass through the common duct by Christian Science or any other sort of remedy that you can induce the patient to take, and therefore operation is in line for that person.

Indications for operation are just those that I have gone over: empyema; make your diagnosis on account of the prolonged period of disease which you see the patient in, the ups and downs, respites in temperature, step-ladder temperature, enlargement, continuous pain in the right upper quadrant, on pressure in particular, that patient has an empyema in all probability or pus in the gallbladder and should be operated upon.

Gangrene has a slightly different appearance in as far as its history is concerned, and gangrene is accompanied as a rule by a high blood count and a very exquisite tenderness on pressure. You can almost invariably make a diagnosis of gangrene from the resistance or the extreme pain that the patient manifests or evidences.

Temperature and blood count of course are there in the majority of instances excepting where the empyema has gone on for some time and the patient has become more or less accustomed to it and you find the low blood count as compared to the other.

What is your choice of operation, limiting yourself to the gallbladder, cholecystectomy or cholecystostomy? Cholecystectomy, gentlemen, every time. I have been hammering at this question of cholecystectomy ever since 1902 or 1903, when I wrote my first article on it that I can recall, and since 1909 when I reported a series of twenty-seven cholecystectomies, a small number in the hands of modern operators, but when we have one case and discuss a paper in the medical meeting we always cite our one case. My twenty-seven cases were then considered quite a few, and it was some hard stepping in New York City to induce the men to do cholecystectomies instead of cholecystostomies. If they did cholecystectomies, they did them in a two-step operation. Two-step operations subject the patient, any patient, to two chances of death, and as far as the question of economics is concerned, you all know what it costs to lie in a hospital even if you are a doctor and get a percentage off.

Why do I throw out cholecystostomy? Cholecystostomy is the opening of the gallbladder and making it drain through the abdominal wall, waiting for something else to happen, either for the gallbladder to close up its sinus or the sinus to be closed up, or the fistula, or to get another attack which requires you to enter the abdomen and clean it out, or he or she disappears from your care and gets in the hands of someone else in the course of a few years.

Cholecystostomy is likely to be followed—let's get this—by a fistula in many instances. Why? Because in the majority of instances where cholecystostomy is done there is a stone left down at the orifice of the cystic duct or in the cystic duct. What happens after that? The gallbladder begins to heal up and then you have a closure of your abdominal fistula and you say, "Oh, what a man I am! I have done a cholecystostomy," and then in a few days or weeks the patient calls you up and says, "I have pain in my abdomen right under my scar." You examine him, and somewhere in the middle of the scar there is a little red elevation, and if you puncture that with a knife or scissors, out come mucus and pus and in all probability some small granular types of stones. Then you subject your patient to the second operation and give him two chances to die of something that he had only one chance to die of before. Mark you, I don't say that a cholecystectomy must be done in every instance; that would be fallacious, that would be exceedingly wrong. Once in a very great while, and I repeat once in a *very great* while, you get a patient so feeble at the time that you do a drainage on him and then subsequently follow it up, if your patient reacts properly, or give him a chance to see whether or not there are any stones left in the gallbladder.

Subsequent to this there may be a fistula following cholecystectomy, and believe me, this is a dangerous thing for all of us to do, especially if we get a little bit theatrical when we are doing work before an audience. The danger to the common duct is a very grave one, and to the hepaticus; the danger to the cystic artery is also a grave one. I believe that numbers of these so-called early liver deaths in the first thirty-six hours are due to injury to the main blood supply of the cystic artery and the hepaticus. We have here two cases with fistula, and we are talking about cholecystectomies that are productive of fistulae, very often, not because of the operation, but because of what was done during the operation. We are either doing grandstand work and snap at the duct—I did my first one that way, I am willing to admit—or we have an anomalous source to the cystic duct, and there are three varieties of these anomalous ducts. One is that which winds about in a spiral, and winds about the common duct or the hepaticus at the junction with the common duct; another is the parallel one which is bound to the duct itself by means of adhesion, or in truly anatomical fashion; and a third one is that where you have a very short cystic duct, and believe me, when you have a short cystic duct you have your hands full.

As far as the cholangiograph is concerned, I will agree with the speakers who have been here in the last two days in reference to it. I do not use the cholangiograph unless there is a definite indication of discharge or repeated or prolonged discharge and I have a T-tube; I do not do the operative cholangiograph because I feel that my fingers and a probe can be put into or under the common duct after we have opened it and afford pretty good tactile sensation. I never have seen a case such as the doctor reported here where I could not put an instrument through the opening in the ampulla of Vater. I suppose my time is coming when it will require some operating-table-side ingenuity to get away from it. You can't sit down and think it over the night before.

We spoke about the question of malignancy and I should like just to add this. What are you going to do when you come up against malignancy? (Slide) Papilloma of the gallbladder will simulate stones in the gallbladder at times by pain, but the x-ray will not show you their shadows in different points of the gallbladder as in stones, but will show you the shadow at one specific point every time. This was a young man twenty-three years of age who had been complaining of some pain in his right side for six or seven years. At the time of examination in 1931 there was nothing demonstrable. In 1934 he again consulted me, stating that he had had bad pain on the right side. X-rays taken showed the shadows.

(Slide) Every single set of pictures we had taken of that man put the shadow at that point; it is always at that point. I did not operate on this gentleman. He is still alive. He is in his thirty-fourth or thirty-fifth year and has had little or no evidence of pain. Have I done wrong in not operating upon a man with papilloma, because all papilloma of the bladder, stomach, intestine, and so forth, have possibilities of becoming malignant?

(Slide) This is a tree of the liver in a woman in whom we operated three times. She had a common duct at the time I operated first, that you could stick your index finger in without any difficulty. Then she got a pancreatitis and hardening of the pancreas itself. This was five or six years ago. Then we reopened her and the duct was enlarged and I could pass a probe beginning with a 2 mm. up to 8 mm. without any difficulty at all through this. I thought the best thing I could do would be to associate that common duct with the jejunum or with the duodenum, which I did, and she went on beautifully, and then about three years after that at least I saw her with another beginning jaundice; I reopened her and found she had without any question a pancreatic malignancy. Whether that was there in the beginning or not I do not know.

Cancer. Remember, I told you that cancers of the gallbladder are said to occur as a result of stones. This patient had several stones in the gallbladder and has a malignancy at this point. I have had three common and hepatic duct malignancies, and you can understand there is very little if anything to do for them when they are hidden in the liver tissue.

(Slide) This is a patient seen by me in 1926 with irregular hard mass in the upper right quadrant. She had lost about ten pounds. She had a positive history of gallbladder colics but no jaundice. Operation revealed carcinoma of the gallbladder with carcinoma of the adjoining liver tissue. This was excised. There was metastasis into the liver. She made a nice postoperative recovery and lived the best part of a year and a half or two years.

(Slide) This was an interesting case of a woman who had all the manifestations of a cholelithiasis and more than likely of the duct. On one X-ray exposure we saw a large mass of gall stones. We did not see any in the duct and I took six out of the duct, as nearly as I can remember, and as I was extruding these stones from the duct a large red body about an inch and a half long and fully a half inch wide popped out which was a papilloma attached to the duct just above the papilla of Vater. I removed that without any trouble, enucleated it, scraped it with my fingernail, found no infiltration around about it, and yet in six months' time this woman came in with exquisite tenderness in her clavicle. We never could get a biopsy from her and never got an autopsy. She died sometime after, I think without any question, of metastases.

(Slide) This patient had carcinoma of the gallbladder. There is a beautiful specimen. Look at the stones in here, and that Christmas-tree-like bulk is a papilloma with its attachment at this point, papillary adenocarcinoma of the gallbladder. She made a beautiful recovery and we followed her for a year or two, and that is the last we knew of her. Possibly she has died since then.

I have one sarcoma here. I don't know how many have been found in the last eight or ten years, but I looked up the literature on the subject at the time I did this case a few years ago, and then there were thirteen sarcomata of the gallbladder operated upon, but twenty-one or twenty-seven had been found between operation and autopsy studies. She had a large tumor in her upper right quadrant and I just took it for an ordinary, every-day dilated gallbladder with an inflammatory condition. At the time I got in there, this is what we found, massively dilated vessels covering over an enlarged gallbladder as large as one of the largest oranges one sees, with infiltration in the portion of the colonic mesentery.

On removal, which was an easy job, we cut off the adhesions of the colon to the gallbladder without any difficulty, and we found five stones in the gallbladder and these purplish masses here. There was an immensely thickened gallbladder. This is the cavity and here is the wall. These are areas of pronounced sarcoma.

The Management of Ruptured Intervertebral Discs

By JAMES L. POPPEN, M. D.
Lahey Clinic, Boston, Massachusetts

The subject of herniated intervertebral disc is now well known, although there has been considerable discrepancy of opinion concerning who should have the credit for first making us acquainted with the subject.

Schmorl acquainted us with the pathology, physiology and anatomy. Then Goldthwait, of Boston, in 1911 became so suspicious that a ruptured intervertebral disc was present that he asked Cushing to do a laminectomy. However, a herniated disc was not found at that time. Dandy, of Baltimore, I believe, should have all the credit for accurately identifying and describing the condition, that is, as a degenerated fibrocartilage due to injury. Certainly, the person who makes us acquainted with a particular disease should have just as much credit as the one who discovers it, and for this we are indebted to Mixter, of Boston. By means of a contrast medium, namely, lipiodol, he first was able to detect the condition early, and in that way, since 1933, many thousands of ruptured intervertebral discs have been discovered and operated upon successfully.

The treatment of herniated intervertebral disc probably will remain a subject for argument from the point of view of whether the bone and joint surgeon or the neurosurgeon should take care of these cases. At the present time I think there is a healthy attitude in that the bone and joint surgeon takes care of the bone part, whereas the neurosurgeon removes the ruptured cartilage. Unfortunately, some suspected herniated discs prove to be tumors, and then, of course, the condition should be taken care of by the neurosurgeon.

In any condition, what happens after operation is important, and I have followed 200 cases closely for three or more years. At the Clinic we have operated upon well over 500 patients. However, one medical group has done over 2,000. Because of our lesser number, perhaps I should not be here talking about the herniated disc. However, these 200 carefully followed cases, I believe, will give us some idea as to the subjective and objective symptoms. For that reason, I would like to show the following slides.

As you see (slide), the males predominate (75 per cent males, 25 per cent females). This is to be expected because the male, as a rule, does heavier work, excepting in the last year or two when the female probably has been, or will be getting, more herniated discs because of participation in heavy war work.

There is a history of injury in 85 per cent of cases. It is important to note that injury rarely is severe with crushing of the vertebra. I like to compare the herniated disc with the subdural hematoma. In the latter the patient rarely has a severe head injury but rather what we call a trivial injury. With a herniated disc, the injury is usually of a minor nature, although it is a rather severe lifting strain, or rather, repeated lifting strains. The injury occurs most frequently in the active group, 58 per cent occurring between the ages of 20 and 40. Whether these persons have inherently weakened discs, I am unable to say.

Ninety-five per cent of these patients have back pain and sciatic radiation. Sixty-three per cent have unilateral radiation and 37 per cent bilateral. Most of the patients complain of paresthesias and some weakness of the extremities. However, a person who has severe pain does not care to move his extremities, and therefore the weakness may be from disuse. The only weakness that really amounts to anything is the actual paralysis and foot drop. In 52 per cent there is radiation of pain on coughing and sneezing.

The weight is unimportant. As you all know, most of the persons weigh between 150 and 200 pounds. Constipation occurs in 47 per cent, but this may be due to the fact that the patient dislikes to strain because of pain. Twenty-five per cent have minor bladder difficulties.

In the objective findings, 90 per cent have a positive straight leg test. By a positive straight leg test, I do not mean keeping the leg straight and putting it at right angles, with resulting pain or tightness. I mean a 40 to 60 degree angle with radiation of pain down the leg or pain in the back. This test may be of help in demonstrating the location of the disc. As you know, anything above the fourth or the fifth lumbar probably will not give a positive straight leg test. However, if the patient has pain in the straight leg test, say to him, "Now I will gently raise the leg a little higher, and you tell me where the pain goes." The pain should go down the back of the leg, down the lateral surface of the calf. Then on lifting the leg slightly higher, gently, the pain usually goes into the lateral or the inner surface of the foot and the great toe. If the pain is in the small toes, one assumes that the disc is at the fifth lumbar space. If the pain goes into the large toe or the inner surface of the foot, one assumes that the disc is at the fourth lumbar space. This rule is not infallible, but it is helpful.

Seventy-five per cent of patients have spasm of the back muscles. Forty-two per cent have sensory changes. Thirty per cent have a list of the spine away from the lesion, and curiously enough some have a list toward the lesion. Twenty-five per cent have fibrillary twitchings. The total protein means nothing; however, if it is elevated, it is, of course, some assurance that the patient has a disc.

A word of caution should be interjected as far as the positive signs are concerned. If the condition is in a quiescent state, a positive straight leg test, spasm of back muscles, or sensory changes may be lacking. There may be no list of the spine or absent Achilles. However, I am willing to say that any patient who has a disc at the fifth lumbar space has an absent or diminished Achilles. At the fourth lumbar space, it may be present even in the acute stage.

Another interesting thing is the number of these patients who have discs and who come originally to the Clinic saying, "I have a low back pain," or "I have pain in my leg with a back pain." In 5,000 such cases, 5 per cent had herniated discs. However, the majority of patients have to be suspected of having malignancy, osteoarthritis, cauda equina tumor, diabetic radiculitis, neuritis, and even herpes zoster.

When a patient says, "Doctor, I have a herniated disc. I have had attacks once in a while in the past twenty years. Now, what can happen to me if I don't have this condition taken care of?" I used to reply, "Well, really, I don't think anything can happen to you except the periodic discomfort." However, I told one patient this, and that night he slipped on the stairs at home and couldn't get up because of a bilateral foot-drop. He also had paralysis of the bladder and of his bowel. He came to the hospital immediately, and was found to have extruded this ruptured cartilage completely into the spinal canal. We have had four such cases. One was the patient of an osteopath who had given him complete relief every time before and then he administered what I call an overactive treatment, and the patient could not get off the table because of a bilateral foot-drop. So now I tell the patient the possibilities. As a matter of fact, it scares most of them, which is unfortunate, but nevertheless I do think they should know the facts.

As far as the diagnosis is concerned, with this typical syndrome of subjective and objective findings, I agree with Dandy in the statement that the spinogram can be dispensed with and the patient operated on, providing the roentgenogram of the lumbosacral spine is normal. However, we do an oxygen spinogram in most cases, because it is a little better to be able to say to the patient, "You have a ruptured intervertebral disc. If it is removed, you have an 85 per cent chance of getting satisfactory relief." Then, after operation you need not say, "I'm sorry, I opened your back and found nothing."

I think the oxygen spinogram is a "must" in a certain group. The patient who does not fulfill all the criteria of a herniated disc should have an oxygen spinogram. The one on whom a fusion is contemplated should have a spinogram to confirm the presence of a herniated intervertebral disc. In a patient who seeks medical attention after two weeks of severe sciatica, it may be difficult to decide whether surgical or medical treatment is advisable. If a large herniated intervertebral disc is found on the spinogram, conservative measures will be of no value. On the other hand, if the herniated disc is small with practically no change in the spinogram, conservative measures may be instituted.

We perform the oxygen spinogram by inserting one needle, taking out 25 cc. of fluid, with the patient in the Fowler's position. We then attach a 50 cc. syringe of oxygen, and then when the patient is in the Trendelenburg position, we insert 45 to 50 cc. of oxygen. However, at times one can inject only 35 cc. It is important to distend the spinal canal, so that the nerve root canals are filled.

Unfortunately, an oxygen spinogram is not dramatic on the screen. I think, however, that this slide shows the outline of the spinal canal. When the bones and joints are normal, with a typical history of a herniated disc, there is an added probability of a ruptured cartilage. Here, however, one can see a large defect in the air column (slide).

Here the nerve root does not fill (slide). It fills perfectly elsewhere.

This demonstrates the spinal canal and the large defect posteriorly (slide).

The lateral views usually show nothing and mean nothing unless a large defect is present (slide).

This slide shows a perfectly normal posterior longitudinal ligament, but a small defect in this area.

On the other hand, with lipiodol the result is absolutely dramatic, and in this slide the defect is obvious. Lipiodol cannot be removed in all cases; however, Hampton and Kubik, of Boston, have been able to inject lipiodol and remove it. In a radiculitis, little meshes of arachnoid prevent its complete removal, and in a few cases it does cause symptoms if allowed to remain in the canal.

Formerly, we did a large laminectomy, but of course that is unnecessary. This slide shows the protrusion with the cauda equina pushed to one side, the nerve root with the little glistening adhesions around it, and the fusiform root demonstrating a localized radiculitis and arachnoiditis. The dura was then opened as well as the posterior longitudinal ligament to remove the degenerated cartilage. By the way, those patients did very well, as we look back on them several years later.

At the present time, however, we make a small incision which is anywhere from 2 to 4 inches in length, and then do a unilateral exposure. This slide shows the muscle stripped from one side. Only a small portion of the inferior border of the lamina has been removed. The ligamentum flavum is incised and reflected laterally. As the wedge is incised laterally, it is displaced medially and an incision made in the lateral gutter. Because of the small opening, it is necessary to have the ligament removed. This slide shows a cross section of where the knife or the scalpel goes and how the nerve root is impinged. Here are the rupture of the cartilage, the ligamentum flavum, and the compressed nerve root.

I doubt the existence of hypertrophy of the ligamentum flavum. I can see, however, how a thickened ligament or a fibrosed ligament is capable of producing pressure on the nerve if rupture of the cartilage exists. Therefore, removal of the thickened ligament many times gives the patient complete relief. Here the forceps is inserted (slide). The nerve root is slightly displaced, and then the whole dural canal is displaced laterally.

To Mixter again belongs the credit for exposing the ruptured cartilage extradurally. To Semmes belongs the credit of doing it through a unilateral exposure. Love, at the Mayo Clinic, has done it without removing any bone whatsoever, and this is possible in patients who have a wide interspace.

Here is the protrusion (slide). The posterior longitudinal ligament is incised. Many times there is rather furious epidural bleeding. Those of you who have done this operation know that epidural bleeding is annoying, but it can be controlled completely by inserting a pledget of cotton in the lateral gutter above and below, thus compressing the veins in the epidural space. After the ligament is incised the cartilage can be pulled out, and the nerve root usually is in the normal position. Removal of the fragmented portion is not enough. All the degenerated portion should and can be removed with a pituitary rongeur.

A few of these patients have a posterior spur (slide). Formerly I excised these spurs with a chisel and in two or three years they grew back as large as before. Now by gouging out some bone from the bodies of the vertebrae and making a compression fracture, a complete decompression can be done.

Only 9 per cent of our patients needed fusion.

Postoperative results. Forty-five per cent of patients were back at work in two or three months; 35 per cent in three to six months; 10 per cent in six months to a year; and 10 per cent in one to two years. One had a complete paraplegia, and it was five years before he could return to work.

Summary. Complete relief occurred in 65 per cent of patients, and 75 to 90 per cent relief in 20 per cent. In other words, satisfactory results were obtained in 85 per cent of cases. The sciatica disappeared in 10 per cent; no relief was obtained in 5 per cent. I cannot tell you why some of these patients did not obtain relief. They had what we call good ruptured intervertebral discs,—none that we manufactured ourselves.

Conclusions. There is such a thing as a herniated intervertebral disc, which can be treated satisfactorily. The symptomatology follows a typical pattern subjectively. Objectively, the findings depend on whether the condition is in the acute or quiescent state. The difficult problem in herniated disc is to decide whether to undertake fusion. I personally feel that a person who has an unstable lumbosacral joint, who does hard physical labor and has reaction around the joint, should be fused when the herniated disc is removed. Other persons should have simple removal of the herniated disc.

MEMORIAM

Wednesday Afternoon, October 27, 1943

Dr. Clifford U. Collins, Peoria, Illinois, Former Trustee
and
Dr. Clarence B. Ripley, Galesburg, Illinois, Former Trustee

ORGAN PRELUDE

TRIBUTE TO DR. CLIFFORD U. COLLINS, BY DR. JOHN M. DODD, ASHLAND, WISCONSIN, TRUSTEE

Life is short; its tenure uncertain; its end inevitable. Man spends the years allotted to him in the pursuit of his ideals, whether selfish or altruistic. If the latter, as Doctors of Medicine are, he devotes his energies to service to his generation and to humanity in general. He is governed by heredity and environment, and by the lessons of the past, applying them to the needs of the present, with the hope that the future of the world and its people will be better for his having lived.

Living in obscurity or amid the plaudits of the multitude he runs his course, has his share of joys and sorrows, enjoys the fellowship of kindred spirits, does his work, fulfills his mission, and then folds his hands and takes his place in the silent halls of death where countless numbers have preceded him.

> "*Strange is it not that of all the myriads who*
> *Before us have the gates of death passed through*
> *Not one returns to tell us of the way*
> *Which to discover we must travel too.*"

Since our last meeting we have bidden farewell to an honored member, a trustee and a Past President, of this association, Dr. Clifford U. Collins of Peoria, Illinois.

Dr. Collins was born at Batavia, Ohio, in 1867, and was 75 at the time of his death, May 26th, 1943. He graduated from the High School at Vandalia, Illinois, in 1884, and in medicine from the Marion Sims College of Medicine, St. Louis, Missouri, in 1892.

He practiced continuously in Peoria since 1904. He was a progressive doctor, specializing in surgery, ever seeking the best in knowledge and practice, organizing a clinic the better to serve his clientele, and to train those who were to follow him in his chosen field.

He was a member and Past President of the Peoria Medical Society. A member of the A. M. A., of the Western Surgical Society, a fellow in the American College of Surgeons and a founder of this Association.

He was active in civic life. Was a Past President of the Peoria Association of Commerce, a Past President of the Peoria Rotary Club, passed through the various degrees of Masonry up to and including the 32nd degree.

Dr. Collins lived a full life and his varied activities attested to his aims in life and his desire to be of service to his fellow men. He lived through and was active in the most wonderful period in the world's history, where Medicine and Surgery kept pace with the advances of science and art and human betterment. He will be missed by a host of friends who knew him. His life will be an example and an inspiration to all who aim at the better things.

TRIBUTE TO DR. CLARENCE B. RIPLEY, BY DR. GEORGE V. I. BROWN, MILWAUKEE, WISCONSIN, TRUSTEE

I deeply appreciate the honor of expressing for the Managing Director and all the Trustees of the Inter-State Postgraduate Medical Association profound regret for the loss of a valued member of our Official Family, whom death has taken from us.

Dr. Clarence Bingham Ripley, who passed away on May 6th, 1943, had shared our meetings only a comparatively short time, but in that brief period demonstrated the value of his advice, and established a warm feeling of personal friendship that made us look forward to future co-operation with much pleasurable anticipation.

He represented a high type in the appraisal of medical and surgical practitioners.

Dr. Ripley graduated from the University of Michigan in 1902, receiving liberal arts and medical degrees. Even in the days of his earliest practice he took part, as he did all his life, in aiding civic institutions. He served both county and city physician assignments, was closely related to the advancement of Galesburg Cottage Hospital, and was Chief of the Staff at the time of his death.

He was physician and surgeon of the Burlington Railroad, and in that capacity was sent to different important situations until 1906, when his growing practice required him to give up that position.

During World War I, he served as Captain in the Medical Corps, was stationed briefly at Camp Greenleaf, and Fort Oglethorpe, Ga., before he was transferred to Camp Meade, Md., and later was assigned to the Evacuation Hospital, and after that to a debarkation hospital, following which he was appointed Assistant Chief Surgeon of the General Hospital at Fort Snelling, Minnesota.

He did his share as a member of the county, state, and the American Medical Association, also as a Fellow of the American College of Surgeons.

In consideration of his outstanding record of professional attainments and his personal attributes, he was chosen to be a Trustee of the Inter-State Postgraduate Medical Association, and his good fellowship amply demonstrated the wisdom of the choice.

Appointment of Trustees to the Inter-State Postgraduate Medical Association is practically in effect for life. Choice in appointment of Trustees depends not only on professional standing, but also on personal acceptability to members of the Board, and in addition an unquestionably good reputation as a citizen.

Because of the sincere regard in which Dr. Ripley was held in Galesburg, his election as Trustee was greeted generally through the press and otherwise as an honor for his home city as well as for personal reasons.

Dr. Ripley was sincere and helpful in his religious activities in the Central Congregational Church at Galesburg which he attended, and also was a member of the Galesburg and Soangetaha Clubs.

Honored for his professional attainments, praised and deeply respected by his fellow townsmen, happy in the affection of a loving wife, gratified by the success of his son now grown to manhood and settled in life, he deserves to have it said in words of praise that have come down to us from the highest of all sources "well done thou good and faithful servant" in the field of human endeavor.

The Assembly stood in silent tribute to the memory
of Dr. George W. Crile, Cleveland, Ohio, and
Dr. Lewellys F. Barker, Baltimore, Maryland.

ORGAN RECESSIONAL

The Treatment of Gastric and Duodenal Ulcer

By GEORGE B. EUSTERMAN, M. D.
Professor of Medicine, University of Minnesota Graduate School of Medicine,
Mayo Clinic, Rochester, Minnesota

I think it is unnecessary to tell an audience of this caliber of the importance of chronic peptic ulcer, not only in time of war but in time of peace. The importance of the subject has been brought home forcefully to us if we review our current articles on military medicine; but not only that, the repercussions of war on the civil population have been very extensive. I do not recall any time in the history of my thirty-five years' connection with the clinic in which so many patients sought us for help, as they do you, because of the strains and worries and anxieties brought on by the war. Many patients who previously enjoyed pretty good health in spite of their ulcer now are having considerable trouble.

In addition to this important aspect of the ulcer problem, ulcer is really in its largest sense a medical problem, that is, one of medical treatment. On that account, we should take particular pains in making ourselves better equipped in the medical management of this disorder.

A number of years ago I was barnstorming, so to speak, with the late and lamented Dr. John Deaver in various cities of Canada, and our discussions were on the medical and surgical problems of duodenal ulcer. This was some twenty-five years ago, and at that time the medical man was hardly tolerated as regards the non-surgical treatment of ulcer. But times have changed. I think the reason they have changed is that in the old days when my surgical colleagues made such an enviable reputation in the surgical treatment of ulcer, we were dealing with chronic, long-standing, complicated cases. As objective methods of diagnosis became more exacting, more of these lesions were found at an earlier age and in uncomplicated states. When surgery of those cases was carried out we began to have our failures, our repercussions, so that surgery of uncomplicated gastric and duodenal ulcer had a sort of temporary setback. I think most of the surgeons see the problem eye to eye, and with one exception which I shall briefly discuss later, I think we are more or less agreed as to the type of patient that should be operated upon and the type of patient that should be treated conservatively.

I had the happy experience of close association with the surgeon. I was not set aloof, by myself, as are most internists in this field, but I had to rub shoulder to shoulder with the surgeon every day in the discussion and the settling of these problems.

Peptic ulcer with its congener, chronic duodenitis, is an extremely common disease. One should not quote figures, but we see as high as 3,000 patients a year with chronic ulcer of the stomach and duodenum and chronic duodenitis, so it is an extremely important problem.

The gentlemen in charge of the program did not ask me to spend too much time on matters of individual details of technic, and I shall not take up too much of your time with those. I am going to discuss more the principles underlying the medical treatment of the disease.

I might say briefly that in the treatment of the uncomplicated gastric and duodenal ulcer, duodenitis and hypertrophic gastritis, we follow in large measure the lines laid down by the late Dr. Sippy, the antacid form of therapy. Of course, we have made a good many variations of the treatment. We have gotten away from the soluble antacids. We have gotten away from the large doses of these alkalis; we have tended to treat the patient as an individual, individualizing our treatment; we go more to the safer antacids such as calcium carbonate, magnesium oxide, and particularly the colloidal aluminum salts, magnesium trisilicate, and the tribasic phosphates. In that way, all types of patients can be treated and treated thoroughly without any fear of that former bugaboo alkalosis. We still encounter alkalosis in the severely obstructed cases. The condition must be recognized and treated beforehand, particularly if operation is undertaken.

I could spend most of the time allotted me this afternoon on the discussion of the differential diagnosis of gastric ulcer and gastric cancer. It is a subject I shall briefly touch as I show these slides, because I want to emphasize the importance of following out closely and carefully every patient with an ulcerating lesion of the stomach, because ten or twelve per cent of these ulcers are actually malignant. There is no way we can recognize it except by histological examination or by the nature of response to treatment.

I might also add that the medical treatment of the ulcer is very adequate in the waking hours. In the sleeping hours we now resort to the method adopted by some of my Crile Clinic colleagues, of awakening the patients at midnight and at two and four in the morning to give them their antacid and their alimentation, if necessary, thereby getting away from that method of jejunal feeding which is very effective but which we do not find essential except in a few chosen refractory cases.

I think we should also remember two important things. In the active ulcer that you wish to heal, the earlier you get the patient the more likely you are to heal him. That is where you men on the front line have the golden opportunity. If you wish actually to heal the ulcer, the patient should be hospitalized for at least three or four weeks. Treatment should be undertaken from seven in the morning until seven at night, hourly feedings, four ounces of milk and twenty per cent cream, the antacids in the interval, the antispasmodics and sedations which are important especially in the first few weeks of treatment. Of course, the removal of foci, the resting of the patient, getting away from emotional tension, avoidance of tobacco, alcohol, and so forth, are primarily essential in the healing of the lesion.

I wish to point out one other thing, that the lesion may be readily healed, particularly the early gastric ulcer, which by and large is less refractory than the average duodenal ulcer; but the big problem is not in the healing of these early uncomplicated lesions but in keeping them healed. Here is where you must cultivate the confidence of the patient, explain to him what you are trying to do, and in that way you are able to have him carry on for a year or longer, as long as necessary, after his release from the hospital, and insure permanent healing in many of the patients. The problem, therefore, in ulcer management is not so much the healing of the lesion as the problem of keeping it healed, and therein you must cultivate the patient's complete sustained cooperation, otherwise recurrence is frequent.

I will close the discussion by showing some slides.

(Slide) The duodenal lesion is usually not larger than the end of a lead pencil. The tendency to healing shortens the long diameter of the duodenum and produces this pocketing, giving rise to diverticula, which explains the clover-leaf deformity which you see on the x-ray.

(Slide) That is a gross specimen showing a lesion in the pre-pyloric area, and you see the smaller area there where there had been bleeding from an artery in the ulcer. These pre-pyloric ulcers are always looked on with suspicion, although my roentgenologic colleague, Dr. Kirklin, has lately shown that all pre-pyloric ulcers with a characteristic history, chemistry, and x-ray deformity are as likely to be benign as ulcers elsewhere in the stomach; but if they have any of the earmarks clinically, chemically or radiologically of a malignancy, then we should not tarry too long with ulcer in that location. That applies also to ulcer on the greater curvature.

(Slide) I brought this film along because there is a good deal of controversy about the size of the ulcer in relation to malignancy. It has been shown statistically that ulcers the size of a quarter or larger in a great many cases are malignant. We have followed that rule and we have been more or less fortunate in that respect. But here is one of the largest ulcers we have had, at operation a few days ago, (slide) it was 10 cm. in diameter. It had perforated the pancreas and the liver and yet was a benign, subacute ulcer, so there are many exceptions to the rule regarding size in relation to malignancy.

It has been said that ulcer of the duodenum and stomach are increasing. I would like to say that as yet we have no statistical proof for that fact because we have not gone about to find the proof. The statistics from institutions like the Clinic or the larger hospitals do not give a clear picture as to the actual fact because the sampling is not correct. Recently a prominent New York surgeon said that the ulcers were increasing in the general population. He quoted statistics from the Clinic which correspond to this area here. If we had accepted that we would have said that his statement is true, but if you follow the cases long enough you find out that you have these up-and-down curves, therefore giving you misleading ideas if you draw your conclusions at any particular ascent in the curve.

(Slide) To show you that ulcers of the stomach and duodenum are largely a medical problem, I want to quote to you from surgical statistics of Peter Bent Brigham Hospital. From 1913 to 1926 the black section represents the surgical group and the cross-hatched section represents the medical, and you notice that from there on there has been a great preponderance of medical over surgical treatment of peptic ulcer in the East. In the June number of S. G. & O., the Lahey Clinic reported 7,000 cases, of which 6,550 were duodenal ulcers and 450 were gastric. Eighteen per cent of the gastric ulcers were operated on and 6.5 per cent of the duodenal ulcers were operated on, approximating the statistics of the Peter Bent Brigham Hospital.

(Slide) Here is a group from our own Clinic back in 1922, '23 and '24, showing the ratio of the surgical to the medical cases. I also want to point out that gastric ulcer is a most uncommon lesion of the stomach, gastric carcinoma being three times as frequent as gastric ulcer.

(Slide) You will see from this slide the number of duodenal ulcers that have been treated at the Clinic surgically and you see a gradual drop in the curve. That is partly due to the fact that so many of these ulcers are picked up radiologically, that many of the patients have very mild symptoms or their symptoms are of secondary importance, so that there has been a gradual conservatism in the treatment of these ulcers, which is the trend, of course, elsewhere

(Slide) This slide shows something about which I have a little quarrel as an internist. This shows the number of patients that are treated surgically for gastric ulcers, and this is done because of the fact that 10 to 12 per cent of our gastric ulcers are diagnosed as ulcers when they are actually small ulcerating carcinomas. With that club over our heads, so to speak, the surgeons say it is dangerous to treat medically these patients. Well, fortunately the surgical mortality is negligible and it is true that recurrences do obtain in these chronic complicated ulcers treated medically, but I still insist (and this is the only quarrel I have with my surgical colleagues) that the percentage of gastric ulcer operated upon may be a little high. There is one other extenuating circumstance. Many of these cases are chronic complicated cases and they come to us after an average of eight, nine, and ten years of trouble, so you cure the patient at the same time with one procedure more or less permanently and you head off the danger of cancer or actually ablate the cancer which may be unexpectedly present.

(Slide) This is an example of how a condition may simulate a benign lesion. I may add that while the surgeon stresses the fact that 10 per cent of these ulcers diagnosed as benign by the x-ray men are malignant, 26 per cent of the ulcers diagnosed as probably malignant by the radiologists are actually benign, so the surgeon ought to show both sides of the problem when he is stressing the other figures. The following case shows how another condition, hypertrophic gastritis, may simulate malignant ulcers, particularly in the presence of a lack of acid.

(Slide) This is an example where the roentgenologic features were those of a benign lesion but the lesion was actually a high-grade malignant one and quite well within the range of the average diameter of the benign lesion.

I might orient you about diameters by saying that 2.4 centimeters is the diameter of a crater of the size of a quarter and 1.8 is the diameter of a dime.

This is an example from one of Bloomfield's papers showing the importance of the role of acidity in ulceration. That, of course, is one of the things that we as medical men and surgeons strive to reduce or eliminate as the basic principle in the management of these lesions. It is not the whole story, but it is an important factor in the pathogenesis of ulcer.

I might say here briefly that we resort occasionally to duodenal tube feeding in some of the refractory cases, particularly the refractory jejunal ulcers. I think parenteral methods of treatment are all right for the detail man and the drug firms, but they are not so much good for the patient. We resort to the orthodox diet, alkalis plus antispasmodics and sedation in the treatment.

As regards the diet, we have a diet booklet which we will be glad to send to anybody who requests it, which gives you all the details of the diet, of the progressive stages of treatment and the general directions and, which is particularly important, the ambulant regimen after the patient has left the hospital or the clinic. Of course, it is true that many of our patients are not treated in the hospital; they are given ambulant treatment because their condition is mild or they refuse treatment; particularly in these busy days when hospital rooms are scarce and the registration is heavy, we just have to forget about treating a lot of these patients who can temporarily get along without it.

The indications for medical treatment we all know and I have briefly summarized them here. I think psychoneurotic patients are important. In many of these patients I am not sure whether the psychoneurosis or the ulcer is at the bottom of the major part of their trouble, so I hospitalize them and after a week or ten days' observation I am pretty well clarified as to which factor is most prominent. It is amazing how often the psychoneurotic factor is much more prominent than the ulcer and the reason so many of these individuals do not get well even after radical surgery.

(Slide) This is important because when we get the presence of a lesion it is always important to ask these questions. We get a good many duodenal deformities of healed ulcers in patients who have never had symptoms. I have seen such deformities in patients with pernicious anemia and with a characteristic achlorhydria. Naturally, you would not treat a patient of that kind for ulcer in spite of his bulbar deformity. If a complication such as obstruction is present we naturally have to meet such complication by specialized methods during treatment, or as a factor of pre-operative preparation.

The contraindications to conservative treatment I think all my surgical colleagues will agree with me are here: failure of frequent adequate medical attempts; the perforating lesion; the cicatricial decompensated pyloric obstruction; large size of lesion; large perforated chronic lesion adherent to another viscus. I should like to emphasize that type of ulcer. We have that chronic type of duodenal ulcer particularly with radiation to the back which from the standpoint of gastric analysis and x-rays is an uncomplicated ulcer, but the ulcer is refractory and because of its chronicity and the posterior radiation of pain that patient is best treated by surgery, a point I would like to bring out which is not always stressed sufficiently.

(Slide) Factors underlying successful treatment I have mentioned. Like any disease to which human flesh is heir, the earlier you get the case the greater the chances of healing the lesion. We got an increased reactivated secretion in the old days when we used the soluble salts. I bring this out to show you that in many of our cases undergoing exact intensive management in the hospital we frequently aspirate after the first week to see what results we have gotten at neutralization. When neutralization is fairly adequate the prognosis is much better, but you can get healing without neutralization, as everyone agrees.

This is an example of the effect of hemorrhage, and we know, of course, that severe hemorrhage is now associated with what we call an alimentary azotemia, which is often a yardstick of the degree of hemorrhage and also has a prognostic bearing.

This shows you the meniscus sign in the malignant ulcer. That is pathognomonic of carcinoma, but you may get an ulcerative carcinoma without tumefaction and therefore no meniscus sign. Yet the lesion is actually malignant.

(Slide) This happened to be a gardener. He had a duodenal ulcer and a short history; this slide shows the normal cap after treatment.

This shows a large gastric ulcer in an elderly man, a former professor of anatomy, with complete healing. I did not want to treat him. He was irascible, hypertensive, arteriosclerotic; but he had had influenzal pneumonia recently. I had to treat him because he had unresolved patches in his lung. Amazingly enough he went on to complete healing and died two years later from cerebral hemorrhage and we could only find the gastric scar with a microscope at necrospsy.

This is an example of how even all signs may fail. The patient was an elderly man with a two-year history of gastric ulcer, with complete healing gastroscopically and by x-ray, and recurrence here after four months. You note this submucosal area in here. The only reason I had him back, much to his disgust as he was feeling fine, was because he was an elderly man with recent onset of symptoms.

This is a gastroscopic picture of a benign lesion. This is the appearance gastroscopically of the smallest carcinomatous ulcer we have in our series.

This is the gastroscopic appearance of a larger carcinomatous ulcer with that nuance of color of living tissue not seen otherwise; instead of a sharply demarcated border it shades into the tissue; and those nodular areas in the base of the ulcer are significant.

This is a little chart we consult when we have confronting us a man with a gastric lesion which may or may not be carcinomatous. We may want to get some criterion of it before subjecting the patient to intensive therapy or to operation. Long duration favors benignancy. Age thirty years or less favors benignancy. Recent onset in elderly patients favors carcinoma. Irregular syndrome favors carcinoma. Anacidity and obstruction favor carcinoma. Meniscus sign complex is practically pathognomonic for carcinoma. Large niche favors carcinoma, likewise irregular outline, and so on. It is one of the charts we use in differential diagnosis. It is not a decisive, definitive factor, but it is an important one in differentiating a benign ulcerating lesion from a small malignant one.

Rapid Treatment of Early Syphilis

By EVAN W. THOMAS, M. D.
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The pioneering stage in the rapid treatment of early syphilis is about over. Some form of quick treatment is now being used in numerous centers, not only in North and South America but in Europe as well, and now the U. S. Army has accepted it for selected cases. But in spite of this recognition, the rapid treatment of syphilis is still experimental. This means among other things that we are as yet uncertain of the best method of attempting quick "cures" both from the point of view of safety and therapeutic results. Precise and thoroughly documented records must be kept of the history, clinical findings and course of each patient. The blood, urine and spinal fluid must be checked for treatment tolerance as well as for cure of the infection. Follow-up observations must be made over long periods of time, if we are to make the most of our opportunities.

Obviously the stress of war time conditions adds enormously to the difficulties of a good follow-up of patients treated for early syphilis in hospital wards, but even though the ideal is impossible of fulfillment and in some places no prolonged follow-up observation is practical, it is essential that at least some large centers make every effort to observe patients for as long as possible after treatment. With the aid of two full-time follow-up workers we still have about 58 per cent of the patients treated during the first year of our experiments with rapid treatment at Bellevue Hospital under observation. They have now been followed for three years or more and over 70 per cent of all patients have been followed for over six months.

Dangers of Massive Arsenotherapy

When the rapid treatment of early syphilis is confined to chemotherapy alone, arsenical drugs can be administered either by an intravenous drip or multiple injections with a syringe. The arsenical drug of choice is arsenoxide (mapharsen or Phenarsine Hcl.). Many attempts to treat syphilis with massive doses of arsphenamine or neoarsphenamine were made prior to the introduction of the intravenous drip technic by Chargin, Leifer and Hyman but all proved to be too toxic for general acceptance. Great as was the success of Chargin, Leifer and Hyman and great as is our debt to them, had the intravenous drip plan of therapy been confined to the use of neoarsphenamine, in my opinion, it would have met the same fate as older methods of massive arsenotherapy. The incidence of reactions such as peripheral neuritis, exfoliative dermatitis and hepatitis is definitely greater with the older arsphenamines than with arsenoxide.

There is one reaction, however, which has not been avoided even with arsenoxide, i.e., so-called hemorrhagic encephalitis or arsenical encephalopathy. This serious and little understood reaction is the single greatest menace in massive arsenoxide therapy, and until we have learned how to prevent it or check it when it occurs, massive arsenotherapy alone will continue to be appreciably more dangerous than the older and more conservative types of therapy.

So far the incidence of reported deaths among patients treated in a period of from five to ten days with mapharsen has been at least 0.3 per cent. So far as I know all of the deaths have been due to encephalopathy. All things considered the incidence of fatalities has been surprisingly low. Certainly it is low enough to warrant continued experimentation and even adoption of the treatment for patients who will not return regularly for routine treatment over longer periods of time. The time saving factor also has great advantages at present because of the war. In New York at least many selectees are inducted into the army very soon after the completion of the rapid treatment for early syphilis and this plan of therapy has proved a great boon to the merchant marine.

The Intravenous Drip and Multiple Syringe Injections

An excellent review of the various intensive methods of treating early syphilis by Cole, Heisel and Stroud was recently published in the J. A. M. A. The references given in that review describe the various technics used. I will not take the time to describe them in detail here. If the intravenous drip is used, a total dosage of from 1 gm. to 1.4 gm. of mapharsen is needed for the best results in a five-day period of treatment. A similar total dosage is needed if treatment is given by multiple injections with a syringe. With the latter method, individual doses can be divided as the physician chooses within limits. At Bellevue Hospital we started by giving an injection of 0.06 gm. of mapharsen on the morning and evening of each day for ten days. Later we gave an injection of 0.1 gm. twice a day for six days. Schoch and Alexander started by giving a single injection of 0.06 gm. each day for twenty days and later adopted a ten-day plan of therapy with one injection of 0.12 gm. a day. If one chooses, there is no reason why the schedule of treatments should not be divided into three injections a day or even four, provided a total of 1.0 to 1.4 gm. of mapharsen is given within a period of six to twelve days.

In considering the relative merits of the intravenous drip versus multiple injections we must first consider the relative toxicity and therapeutic effects of the two methods and then the relative ease of administration, the cost of treatment and the comfort of the patient during treatment.

No final decision as to the relative toxicity of these two methods can as yet be given. From such reports as have been made, however, there is no significant difference in toxic effects between the two technics, provided the single doses given by syringe do not exceed 0.1 gm. in which case the period of treatment should not be less than six days. The incidence of encephalopathy with multiple injections at Bellevue Hospital was practically identical with that of the intravenous drip method for a similar number of patients treated with mapharsen. We had one death in our series of 275 patients while the Mt. Sinai Hospital group had none in a series of 288 patients treated with the intravenous drip. Toxic effects other than encephalopathy noted in these two series were slightly higher with the intravenous drip than with multiple injections. Eagle and Hogan found no significant difference between the toxic effects noted in rabbits when the intravenous drip was compared with multiple injections, if a similar total dosage of mapharsen was used over the same period of time. They quite properly point out that comparisons between the two methods in man are somewhat complicated by the fact that in the earlier experiments no attempt was made to adjust the dose in accordance with body weight. According to Eagle and Hogan the most satisfactory curative dose of mapharsen in man seems to be about 25 mgs. per kg. which makes the total dosage of mapharsen somewhere between 1.2 and 1.8 gm.

With dosage approximating these amounts, equally good therapeutic results can be expected from either the intravenous drip or multiple syringe methods of administration. This is verified by such statistical reports as are now available in the literature as well as by experiments with rabbits. In rabbits, according to Eagle and Hogan, mapharsen administered by the intravenous drip was usually less effective than mapharsen administered over the same time intervals by repeated injections.

Turning now to the practical considerations of ease of administration, the expense of treatment and the comfort of patients during treatment, there seems to be little doubt that the advantages here lie with repeated injections rather than the intravenous drip. The only exception to this is the additional day of treatment, if multiple injections are given over a six-day period. There is no reason, however, why the multiple injections should not be completed in a five-day period if three or four injections are given each day instead of two. In fact four injections of 0.06 gm. of mapharsen a day for five days might well prove better than two injections of 0.1 gm. a day for six days.

Regardless of which method of administering the drug is used, when an adequate dosage of mapharsen is given over a five or six-day period, the margin of safety is not very great. In fact the incidence of about 1 per cent encephalopathy with 0.3 per cent deaths was so distressing to us at Bellevue Hospital that we began to search for other safer methods of rapid therapy within the first year of our experimentation with rapid treatment for early syphilis. But in spite of this, where the need for speed is great and where the number of days of hospitalization for each patient must be reduced to the smallest practical number, either intravenous drips for five days or repeated injections over a five or six-day period are sufficiently safe to warrant their use in terms of the need. To my mind the preference between the two methods belongs to repeated injections rather than to the intravenous drip for the reasons already given, but it is well that both methods should have further trial before coming to a final decision. The addition of bismuth injections to the five-day treatment with mapharsen has apparently not increased the risk and may enhance the therapeutic effects from present indications.

It is natural that the effort to find the safest plan of rapid treatment should have followed along three different lines. The first was to find the minimum total dosage of arsenoxide consistent with satisfactory therapeutic results. This was approximately determined for the five-day intravenous drip by the investigators directing the experiment at Mt. Sinai

Hospital in New York. Their findings have been confirmed by other investigators so that we know that from 20 to 30 mgs. of arsenoxide per kg. of body weight are required. Having discovered the minimal satisfactory total dosage the next task was to determine the safest period of time over which this could be given in divided doses without loss of "curative" results. The third plan of attack was to find some protective procedures which would prevent serious reactions to arsenoxide or enhance the effect of treatment so that the margin of safety would be increased by permitting smaller doses of toxic drugs. Eagle and Hogan directed their efforts largely to answering the second question and as a result they advise a plan of treatment which does not require hospitalization and lasts over a period of weeks.

EIGHT WEEK PLAN OF MULTIPLE INJECTIONS

Eagle and Hogan were the first to investigate in rabbits the toxic factors of massive arsenoxide therapy in relation to the period of time over which treatment was given. Basing their plan of therapy in man on their experimental work with rabbits, they at first suggested three injections of about 0.06 gm. of mapharsen a week for four to eight weeks. The dosage was roughly approximated to body weight. Clinical experiments in man soon demonstrated that an eight week period of such treatment was superior to shorter periods of time, thus confirming the desirability of a total dosage of from 1.2 to 1.8 gm. of mapharsen. Subsequently Eagle advised the addition of one injection of bismuth subsalicylate in oil each week during the course of the eight weeks.

This is an excellent program of therapy for early syphilis in patients who are reliable and who will attend a physician's office or a clinic regularly for injections. Available data indicate that this method is safer than more concentrated dosage in shorter periods of time. Consequently, it can be recommended to the general practitioner who wishes to treat ambulatory patients who will return regularly for injections at appointed times. For clinics which must treat poor and irresponsible individuals with early syphilis, this plan of therapy has not been practical because of the failure of patients to return regularly for treatment. This has been our experience at Bellevue Hospital where we have had to limit the eight weeks treatment to patients whom we think are responsible and even then we have had difficulties in completing treatment schedules without interruption. It is obvious, however, that for the private practitioner the plan has many advantages.

COMBINED CHEMOTHERAPY AND FEVER

In the effort to find some adjuvant to massive mapharsen therapy which would prevent arsenical encephalopathy, experiments with combined mapharsen and fevers were started at Bellevue Hospital in 1940 following the death of a patient who had been receiving 0.1 gm. of mapharsen twice a day for six days. We hoped that the addition of fever might kill two birds with one stone by protecting patients against the toxic effects of arsenoxide and at the same time enhancing the effects of treatment. Thus we tried various combinations of mapharsen and fevers induced by typhoid vaccine. Sufficiently large numbers of patients were included in each combination to enable us to evaluate toxic effects and therapeutic results. The number of fevers varied in different series from two to four and the total dosage of mapharsen varied from as low as 0.54 gm. to as high as 0.84 gm. The period of time during which patients were treated varied in the different series from seven to ten days. Up to July 15, 1942, each plan of therapy called for two injections of about 0.06 gm. of mapharsen on at least some days of treatment.

In spite of the addition of fever we still encountered about 1 per cent cerebral reactions and by July, 1942, we had two additional deaths to the one already mentioned. It is interesting but not necessarily significant that each of our deaths occurred in very hot weather, although the incidence of encephalopathy was evenly distributed throughout the year. Unlike the experience attributed to Udo Wile reported by Cole et al., we found no relationship between menstruation and encephalopathy, and the incidence of cerebral reactions in our series was actually higher in males than in females.

Following our last fatality in July of last year we studied our records to discover which of the various methods of treatment we had used gave the fewest serious reactions. We found that in a series of 280 patients who had received mapharsen alone in a period of from six to ten days, there were three cases of encephalopathy or a percentage of 1.07, and one death, a percentage of 0.36. Of 588 patients treated with combined mapharsen and fevers induced by typhoid vaccine, eight had encephalopathy, a percentage of 1.36, and two died, a percentage of 0.34. Thus it seemed that the addition of fever had failed to alter the incidence of arsenical encephalopathy. But among the 588 patients treated with fever and mapharsen was one group of 128 patients who had a total dosage of only 0.54 to 0.6 gm. of mapharsen and four fevers induced by typhoid vaccine. One patient in this group had a mild cerebral reaction but he had received two injections of mapharsen a day on two of his treatment days. The results of treatment among the patients followed in this group were by a slight margin the best in the entire series, 86.4 per cent having negative blood and spinal fluid tests over six months after treatment.

With this information in July, 1942, we decided to give no more than 1 injection of from 0.05 to 0.08 gm. of mapharsen on any one day and determined that treatment should be given over a ten-day period with four fevers induced by typhoid vaccine on the 2nd, 4th, 6th and 8th day. Consequently, we outlined the following plan of therapy which is still being used in our wards: Patients weighing from 90 to 110 pounds receive a daily injection of 0.05 gm. of mapharsen for ten days and four fevers induced by typhoid vaccine. Patients weighing between 110 and 155 pounds receive .06 gm. of mapharsen daily, those weighing from 155 to 200 pounds receive .07 gm., and those over 200 pounds receive .08 gm. daily plus four fevers during a ten-day period. In order to prolong the fevers for at least four hours, two injections of typhoid vaccine are usually given two hours apart on the days fever is induced.

This plan of treatment has been followed in the great majority of patients since July 15, 1942. In June of this year we decided to add bismuth, giving 1 cc. of bismuth subsalicylate in oil on the 1st, 4th, 8th and 10th days.

By October 1st of this year we had treated over 800 patients with this plan of combined fever and chemotherapy without a single case of encephalopathy and no deaths. We had one incidence of mild agranulocytosis, the patient recovering with the aid of transfusions. This was our only serious reaction. From our point of view this record is better than we dared hoped for. It has been achieved with a steadily rising rate of admissions and a decrease of nursing facilities. In 1939, we had slightly over 300 admissions with early syphilis to Bellevue Hospital. Since August 1st of this year we have treated over 100 patients a month in the wards of Bellevue Hospital, most of whom received the treatment which I have outlined.

The question arises as to why the incidence of cerebral reactions has dropped so amazingly with this plan of therapy. Possibly the increased caution and wisdom which come with experience have had something to do with this, but the chief explanation seems to lie in the fact that the total dosage of mapharsen is cut in half by this method and only one injection of about 0.06 gm. is given a day. In addition it may be that we were wrong in concluding that fevers had no protective value against reactions from massive arsenotherapy. It is quite possible that some such protective action does occur when four fevers are spaced over the ten-day period of treatment. But the chief value of fever is that it enhances the effectiveness of chemotherapy, thus permitting a lower total dosage of arsenoxide. Apparently it is not the body temperature alone which matters so much as the biologic changes involving the defensive mechanism of the body when fever is induced. This may be true of fever produced by mechanical means as well as with pyrogenic agents such as typhoid vaccine. It is known, for example, that Spirocheta pallida can survive a temperature of even 106° F. for over eight hours, but the induction of fevers in man has a beneficial effect on the disease syphilis, and there is no question that fever greatly enhances the spirocheticidal action of specific drugs.

The chief complication which we have encountered with the ten-day treatment of early syphilis with combined mapharsen injections and fevers is the occasional occurrence of an acute arsenical erythema or the so-called ninth day erythema of Milian. Unfortunately this relatively benign reaction may start on the seventh day of treatment rather than the ninth. It usually begins with a rise in temperature and sometimes is associated with a sore throat and tender cervical nodes. The skin rash may be delayed as long as 48 hours after the fever has started. No arsenical drugs should be given during the course of this reaction and it is desirable to wait at least two weeks before attempting arsenical drugs again. Consequently, the occurrence of this reaction may interrupt the treatment on the seventh day of a ten-day course of therapy. We have not found this a serious handicap because we terminate mapharsen therapy at once when such reactions are noted. It is possible to give the patients additional therapy with bismuth for several weeks and the therapeutic results have proved satisfactory in most cases. Because of the occasional association of encephalopathy with a so-called ninth day erythema, it is advisable to examine the spinal fluids of patients with this reaction for increased protein and cells, if there is the slightest suspicion of cerebral involvement.

In fact our experience with massive arsenotherapy has taught us to depend greatly upon spinal fluid findings in the detection of early and mild cerebral reactions without obvious clinical symptoms. Thus patients who develop unexplained fevers during the course of treatment or who complain of any symptoms which cannot obviously be attributed to the usual effects of the treatment are subjected to spinal fluid examinations. If the fluid shows abnormal increase in protein, arsenical drugs are stopped at once.

An arsenical encephalopathy in our experience gives much higher values for total protein than are usually found in early syphilis of the central nervous system.

CHEMOTHERAPY AND ELECTROPYREXIA

It is impossible at the present time to evaluate the practicability of one or two day periods of treatment for syphilis where chemotherapy is combined with fever induced by mechanical means. Simpson, Kendall and Rose have reported a group of 27 patients treated in one day by this procedure. The brevity of the treatment period has great advantages but at present these are outweighed by the lack of adequate information regarding the best technic to be followed. I am personally convinced from observations of "hot box" therapy of other conditions besides syphilis that sessions of fever over 105° F., prolonged for over five hours, are associated with some risk. In the hands of experts who have had much experience the risk may be negligible but the inexperienced are certain to have difficulties with this treatment, if it is given routinely to large groups of patients.

A two-day treatment consisting of 0.24 to 0.28 gm. of mapharsen in divided doses and seven hours of continuous fever at about 105.8° F., induced by electropyrexia, at Bellevue Hospital, resulted in four cases of acute nephrosis in a series of 48 patients. The treatment may have merit but until further data have been released, those who contemplate this type of therapy must realize the need for special training. From my own inadequate experience, patients must be selected for this type of treatment with considerable care and no one should be persuaded to take it against his will. A well trained personnel is essential for successful care of patients. This means considerable expense but, if one or two days of therapy prove safe and therapeutically satisfactory, the cost of equipment and nursing care may prove economical in the long run. When shorter sessions of not more than five hours of artificially induced fever at about 106° F. are given, we have found very few complications at Bellevue Hospital.

RESULTS OF RAPID TREATMENT

Within the next few years a genuinely comprehensive review of the results of the various rapid methods of treating early syphilis should be possible. At present the most we can say is that in all probability cures are achieved in about 85 per cent of the patients treated with any of the more thoroughly tried methods described in this paper. So far this has been true at Bellevue Hospital and interestingly enough we have found practically the same percentage of probable cures in the series of retreated patients who relapsed or were reinfected. The very high percentage of completely negative spinal fluids examined from one to four years after treatment is especially encouraging.

In the follow-up of patients after treatment it is desirable, if possible, to have titered complement fixation or flocculation tests. As long as the titer is falling there is little need for anxiety, even though it takes six months and occasionally longer before the tests become completely negative. Sudden rises in titers mean trouble and in our experience infectious lesions can usually be found by careful search in most patients with marked increases in titers. An example of this is found in one of our recent relapses. A colored woman with secondary syphilis was admitted to Bellevue Hospital in May, 1943. She had a titer of 180 on admission. By July 16th, the titer had fallen to 29 but on her return in September she had a titer of 140. Examination revealed a small erosion on the vulva which would have been overlooked had it not been for the rise in titer.

TRICHINOSIS

By HOBART A. REIMANN, M. D.
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Trichinosis paradoxically is a rare disease, but a very common infection. By this is meant that about 16 per cent of persons in this country are or have been infected with Trichinella spiralis without being aware of it, and in only a small percentage of them is the infection severe enough to give rise to symptoms. On the basis of the figure 16 per cent, there are estimated to be in the United States about 20,000,000 persons who are infected, representing an estimated 300,000 new cases each year, yet only about 500 cases of trichinosis were reported in 1941. It is certain, therefore, that the actual number of infections of clinical significance is much larger than the number of reported cases for the reason that many patients are not sick enough to seek medical advice, and many that do are, no doubt, misdiagnosed. Physicians who depend wholly on the usual textbook description of the "typical" case of trichinosis to make diagnoses will most often fail to recognize the disease, for in the majority of cases the disease is mild and the symptoms atypical. The so-called "typical" case is the relatively severe form in which there are the so-called "classical signs or symptoms," namely aching muscles, fever, periorbital edema and eosinophilia all occurring at once.

The absence of, the mildness of, or the severity of symptoms depends upon the amount of infected meat eaten and the number of trichinella larvas it contains, on the size and general condition or reaction of the host and upon the presumed hypersensitive state which follows repeated infection.

Clinical

The incubation period is variable. After a heavy infection the onset may be 24 to 48 hours after ingestion; more often the period lasts five to fourteen days or even three weeks. In the early period, during which the larvas are invading the gastrointestinal tract, there are nausea, vomiting and diarrhea; the latter may be severe. In milder cases there may be few or no gastrointestinal symptoms. Malaise, weakness, headache and cough, present in most other infections as well, are common. Cough may appear about the tenth to fifteenth day during the passage of trichinella larvas through the capillaries of the lung. Coughing often leads one to suspect influenza or bronchitis. Coryza, laryngitis or pharyngitis may also occur.

Fever appears from the tenth to the fourteenth days occasionally with chills, sweats and headache. It may reach 105° F., is often intermittent, and declines gradually. Typhoid fever may be suggested at this stage. In mild cases there may be no fever.

Some of the most characteristic symptoms, namely, conjunctival injection, photophobia and later periorbital edema accompany the fever and precede the generalized symptoms caused by the dissemination of the larvas by the blood stream and their deposition in muscles which occurs after the fifteenth day. Ocular symptoms may dominate the picture and cause the patient to consult an ophthalmologist first. Severe pains in the muscles, myalgia and myositis may be generalized, but more often affect certain muscle groups. Pain may be constant or present only on movement. If the diaphragm is invaded, difficulties in breathing resulting from atelectasis and pneumonia may occur; larvas deposited in the heart muscle cause myocarditis.

Confusing skin rashes occur in the form of scarlatiniform macules or ecchymoses. Urticaria which appears in a few cases together with the usual eosinophilia suggests the development of the allergic state against trichina antigen, especially in cases of reinfection.

If the central nervous system is invaded, a variety of confusing symptoms suggesting encephalitis, meningitis, or other conditions may arise. The most serious consequences of the infection as one would expect result from the invasion of the heart, diaphragm, lungs or nervous system. A partial list of the symptoms and signs in trichinosis and an accompanying list of the diseases which may be mistaken for trichinosis during its two stages as prepared by W. H. Wright are shown in the table.

It must be reemphasized that in the majority of cases the infection is mild and the symptoms uncharacteristic, consisting chiefly of weakness, fever, aching muscles, and headache, symptoms which are found in a host of other infections as well.

The prognosis depends upon the number of trichina larvas eaten, upon the condition or resistance of the patient and upon complications. Mild infections may last several days to a week; severe ones

Symptomatology of Trichinosis and the Conditions with which the Disease May be Confused

(Wright, W. H., Journal-Lancet. 62-389, Nov., 1942.)

Stages	Time of Onset After Infection	Location of Parasites	Symptoms which May be Exhibited	Sometimes Tentatively Diagnosed as:
Enteral	24 to 72 Hours	Larvae and Adults in Intestinal Lumen and Villi	Nausea Vomiting Diarrhea Constipation Abdominal pain	Typhoid Fever Food Poisoning Intestinal Influenza Colitis Appendicitis
Parenteral	After 4 to 5 Days	Larvae in Blood Stream and Muscles	Eosinophilia Irregular Hyperpyrexia Edema (especially Suborbital) Conjunctivitis Photophobia Myalgia Sore Throat Dyspnea Cough Scarlatiniform Rash Rose Spots Urticaria Pleurisy Pneumonia	Arthritis Rheumatism Upper Respiratory Infection Laryngitis Conjunctivitis Influenza Intercostal Neuritis Measles Frontal Sinusitis Asthma Pleurisy Pneumonia
		Larvae Passing Through Heart	Chest Pain Tachycardia Apical Murmurs Dicrotic Pulse	Myocarditis Endocarditis
		Larvae in Brain and Meninges	Severe Headache Malaise Marked Hyperpyrexia Disorientation Delirium Coma	Encephalitis Meningitis Poliomyelitis

three to four weeks. The mortality results chiefly from pulmonary, myocardial or neurologic involvement, or from secondary infection, and is said to be 6 per cent of *reported* cases, but certainly is very much lower if calculated on the basis of *all* cases.

Diagnosis

As indicated in the above discussion, it may be impossible to diagnose mild uncharacteristic cases which comprise the majority, unless there is the clue of having recently eaten poorly cooked pork (or bear meat), or if a small epidemic occurs in which "typical" or severe, easily diagnosed cases are also present. Because of the prevalence of trichinosis in so large a percentage of the population, it is desirable in every case of fever, headache, weakness and myalgia to search for eosinophilia. Eosinophilia of over 10 per cent is strong presumptive evidence, but diagnosis must be strengthened by the specific precipitin test, or the skin test. Even these tests are not entirely reliable: one may be positive, the other negative; they may be positive at one time, negative at another; they may both be negative in proved cases, and both or either may be positive during infection caused by other agents as a result of previous infection with trichina or of subclinical trichinosis. Eosinophilia may be absent. The final proof of diagnosis rests on the demonstration of encysted larvas in muscle removed at biopsy. Larvas rarely are found in the blood, feces, spinal fluid or elsewhere. Larvas should not be searched for in muscles before the twentieth day. Even then they may be difficult to find in sections of muscle and can best be seen by pressing the mass of excised tissue between glass plates or by digesting it and examining the residue sediment microscopically. Even with these methods Wright found larvas in only half of a series of known cases. Failure to find larvas at biopsy does not rule out the disease.

TREATMENT

There is no specific remedy. A purgative or an anthelmintic if given in the earliest stages, usually before diagnosis is possible, may serve to clean out many larvas still in the intestines. The sulfonamides should not be used, unless secondary infection with sulfonamide-sensitive bacteria occurs.

The patient should be kept in bed if sick, especially if diaphragmatic, cardiac, pulmonary or cerebral invasion occurs. Analgesic drugs such as aspirin or aminopyrine may be used to relieve the aching, but in severe cases codeine or even morphine may be necessary. The bowels should be kept open and a liberal diet given. Further exposure to infection must be guarded against.

PREVENTION

Since pork is by far the greatest source of infection the simple expedient of thoroughly cooking it, or discarding known infected meat, would prevent infection. Unfortunately, inspection of meat is not always made, and if made may be unreliable. Furthermore, the habit of eating uncooked or partially cooked pork in the form of sausage and otherwise is common. According to present views, the severest infections occur in local small groups of people who eat meat from a single infected animal. On the other hand, in the wholesale preparation of ground meat products or sausage, the meat from a single infected animal is diluted by the meat from many noninfected ones and the subsequent human infection is apt to be mild but far more widespread. Perhaps the best method to prevent trichinosis which strikes at the source of infection would be to prevent infection in hogs by feeding them uninfected food or by feeding them cooked rather than raw garbage.

BREAST FEEDING

By CLIFFORD G. GRULEE, M. D.
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When I was a boy there was a book called Hiram Goff's Religion. Some of you may be old enough to remember that book—most of you won't. Hiram Goff likened the different religious sects to the spokes of a wheel, the hub of which was heaven. He said it didn't make any difference which one of the spokes you took, if you followed it you got to heaven. There is a good deal in infant feeding that is the same way. Nobody can tell you just exactly how to feed a child, and each man has his own ways, each has his own little modifications. In other words, infant feeding is an art and not a science in the true sense of the word.

The question of breast feeding seems to me to be peculiarly live at the present time. One reason is that they are thinking of rationing milk. The one thing they won't ration is breast milk, but since it is unquestionably, I think, the best food, taken by and large, for babies it is very important that we conserve the supply of that breast milk.

I was recently in New York at a meeting, and Charles Hendee Smith, who had just retired as head of the Department of Pediatrics at Bellevue, made the statement that 95 per cent of babies in his new-born wards were breast fed.

I want to tell you a little experience that we had. By a very simple procedure we raised the percentage of breast-fed babies in our wards at the Presbyterian Hospital from something like 48 to almost 90. In other words, I am absolutely persuaded that neither have the women nor the mammary glands of this nation degenerated. I am just as certain that it is the doctors who have degenerated.

I want to give you some slides of a study that was made some ten years ago and to call your attention to the fact that this is a study made on 20,000 babies taken from the Infant Welfare Society here in Chicago. Twenty thousand babies is more than any pediatrician in this country ever saw in a lifetime, and these 20,000 babies were observed over a period of five years. The studies are to compare the advantages and disadvantages of breast feeding and artificial feeding. It should be said that in the Infant Welfare Society in Chicago, of which there are some twenty or twenty-five stations in the poorest districts of the city, there is a very strong effort to see that the mothers nurse their babies and if there is no advantage in breast nursing it should show up in such statistics as we were able to gather at that time. These statistics are somewhat old, but due to the exigencies of war our second compilation of statistics on the same subject has been held back and we have not been able to finish it, so I cannot give you anything later than these, which are from 1925 to 1929. However, during that period everybody had money, these people had plenty of money to buy food and they got it, they had plenty of cod-liver oil and orange juice; it was at a time when we knew enough about vitamins to make it possible for us to give an artificially fed child very good food.

(Slide) This is to show you the nationality of these people. They were just a cross-section of what we find in the poorer sections of a large city in this country. They were picked from these various poor families and they were chosen because they had stayed under the Welfare supervision as long as nine months or had died while they were continuously under its supervision.

This shows the total number of cases. You will see that 48.5 per cent of those children were entirely breast fed for the full nine months. The only exception to that is that they were given some adjuvant food like cereals or vegetables after they were five or six months old, but they were breast fed. You see that practically half of them are breast fed. In the next group, 43 per cent were partially breast fed; that is, for at least a long period of the nine months they were totally breast fed. Then we find that a small percentage of 8.5 were entirely artificially fed. Please remember those figures because they are of a great deal of importance in estimating some of the future figures.

(Slide) That was the percentage of respiratory infections in this group and you see that the breast fed had fewer respiratory infections.

The gastrointestinal infections, as you would expect, were smaller in the breast fed.

The unclassified affections were much higher in the artificially fed than in the breast fed, four times as much, nearly. The total number of infections increased as the breast feeding decreased, almost twice as many in the artificially fed as in the breast fed.

Deaths. Notice that, please. There were only 218 deaths in these 20,000 babies, and of those 218 deaths, 62.1 per cent were in the 8.5 per cent who were artificially fed.

The gastrointestinal deaths were 70 per cent in the artificially fed, although that constituted only 8.5 per cent of the total.

Unclassified, 51.2 per cent as against 12 per cent breast fed.

The total deaths were 218, of which 15 were in the breast fed, 58 in the partially breast fed, and 144 in the artificially fed, and somebody who is quick at figures will add that up and say, "Well, you are one shy." That child burned to death.

This is the monthly morbidity. This is according to the age in months of the child. The breast fed is the straight line and the broken line is the partially breast fed, and the artificially fed shows the increase right up to the ninth month. These are respiratory infections.

These are gastrointestinal. You see here a marked drop in the gastrointestinal infections in those that are partially breast fed, and in the breast fed.

This is the unclassified. It goes up with the artificially fed, right straight through.

I think that demonstrates probably as clearly as one can from a statistical standpoint the benefits derived from breast feeding. All these children were receiving the same type of care and they had just as much supervision one as the other, but the breast fed throughout did much better. That was not on a gastrointestinal basis because those children who were breast fed had fewer respiratory infections, they had fewer infections of a non-gastrointestinal nature other than respiratory. Not only was that true, but from their infections they had far fewer deaths. There were ten times as many deaths in the 8.5 per cent of artificially fed as there were in the 48.5 per cent of breast fed.

I have told you this because there is so marked a tendency so say, "Well, we know it all. We have now arrived at the time when we know all there is to know about nutrition and we can feed a baby artificially just as well as we can at the breast." Gentlemen, that is all poppycock. I have lived for a good many years. I taught at the old Rush Medical College beginning in 1908, and I know that we have thought for a long while that we knew all there was to know about some subject only to be bowled over completely by some new discovery.

We thought we knew all there was to know about it. Then they found vitamins. What they will find next I don't know, but I do know this: that they have not duplicated nature as far as food is concerned, and, like the Scotchman, "I hae me doots" if they ever will.

What is the problem with regard to breast feeding? The truth of the matter is that as long as we were delivering mothers in the home we didn't have the problem of breast feeding to anything like the degree that we have now when we are delivering them in the hospital. Let that sink in because it is true. Why was that? This is the thing that happened to us. We had an in-patient maternity service and we had an out-patient maternity service, and for years we had been looking after the in-patient newborn, that had been part of the pediatrician's job, and then we began some years before this study was made to look after the out-patient newborn. Our interns made calls two or three times during the newborn period. One of my residents who had been keeping pretty good track came to me one day and said, "What do you think of this?" When I looked it over I found that of those out-patient newborn 97 or 98 per cent were breast fed at the end of the lying-in period. I went to the man who was looking after our newborn service in the hospital and said, "What proportion of the children in the hospital are breast fed when they leave the lying-in period?" He said, "I don't know but I will look it up." He was a pretty fast worker and the next day or two he came back to me and said, "Forty-eight per cent."

I said, "How come?"

He said, "I don't know."

I said, "I think I do. I wish you would try it. Try giving those children nothing but the breast for the first week, except water."

He said, "All right, we'll try it."

At the end of about six or eight months those figures changed to approximately 90 per cent.

The stimulation of the breast by the child is the one chief thing to promote in breast feeding. If that child is satisfied with what he is getting some place else, he is just like any other human being in the world, he won't work hard to get what is harder to get. You may think that he is pretty small to have that trait of human nature, but it is absolutely true. If you want to have your children fed on the breast you had better not poke bottles into their mouths while they are newborn. There is not any concoction that anybody in this world has ever developed that will fit those newborn nearly so well as breast milk.

There has been a lot of talk recently about the diarrhea of the newborn. A few years ago they had an epidemic of diarrhea of the newborn in one of the hospitals and made a survey of the conditions and reported them to a committee which Dr. Bundesen called together. I asked one question, and only one: How many of those children were breast fed. None of them was. It is perfectly possible, of course, that a diarrhea in the newborn will develop from another case of diarrhea and still that child may be breast fed. We have had cases of diarrhea of the newborn develop in our hospital, and the only way we were able to save a group of prematures was by giving them breast milk.

I think that goes a long way toward settling two questions. The first is: Is breast milk an advantageous food for children? Second, why is it that the women today are not nursing their babies? They are if they are properly coached.

I do not know who is to blame for this thing. I have heard it blamed on the pediatrician, I have heard it blamed on the obstetrician, I have heard it blamed on the general practitioner, and I have heard it blamed on the nurse. I think it ought to be blamed on human nature. If you stop to analyze the situation you see this: that a person who goes into the hospital usually goes in because he is sick. When a well baby is born in a hospital the attitude toward everything that happens to that baby is the attitude toward a sick individual. If that child cries, it cries because it is sick; if it loses weight, it loses weight because it is sick; if it does a number of different things it does them because it is sick. The natural reaction of human beings is to help somebody, and they do it, but they do not do it judiciously. I think that is the answer to the situation. I do not think it is a question of whether the breast glands are active or whether the mothers are getting enough vitamin this or vitamin that or whether they are getting enough milk. Those things are important, but the basic thing is something much deeper in human nature than that. So my theme would be: Do not poke a bottle of milk into a baby's mouth in the newborn period.

I am far from thinking that you can solve the ills of the world with one formula. I know very well that in any service you will have exceptions to these rules, but I know that in only one instance during my whole experience where I weighed children before and after feeding was I ever able to show no gain in weight, I mean no evidence of breast milk flow from weighing before and after nursing. The woman was very anxious to nurse her baby. She was an old primipara and this was her only child and her only hope of having a child and she wanted to nurse that baby badly and she had not one drop of milk as far as the scales showed.

There are one or two other things that I want to say about breast feeding. I wish I could say them with the expletives that I sometimes feel are necessary for the occasion. Somebody some years back told somebody else and put it into print that you had to nurse a breast baby twenty minutes. I don't know who did it, but I want to say that there have been more babies that have been weaned because of the twenty-minute period than have ever been fed because of it. The reason is that those babies get practically everything they can possibly get in ten minutes. You weigh them before and after and see. There are exceptions, but the average baby does that. The consequence is that it pulls on a dry breast and wears itself out in the other ten minutes. Not only is that true, but it makes the nipples sore and it cracks the nipples.

Very, very many babies will get along very well on five minutes at the breast every four hours, and I think that we have made the mistake of trying to overfeed our babies and that is not alone true in the breast-fed baby; it is also true of the artificially fed baby.

This will seem very peculiar to you when I say it, but our idea of health in a baby is about the way in which we gauge a sausage. The tighter the skin is drawn the healthier the baby and the better the sausage. I can't agree with that. I think that we have gone to far extremes in breast feeding and artificial feeding in cramming these babies full.

There is one other point that I want to make about breast feeding, and that is that the nervous system of the mother is one of the things which must be conserved in all cases. Time and again you see a mother go home from the hospital who may be nursing her baby all right, she has had plenty of help, she has had no worries, and she goes home to a house where she has to look after the rest of the family, she has to bear all the cares of the home and maybe do the cooking, and the consequence is that her breast milk goes because of the worry. Worry is a very potent factor. Remember that the breast gland is a secretory gland and as a secretory gland it is affected by psychologic reactions.

The Heart Attack and Its Prevention by Operation*

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I should like to discuss coronary artery disease and the heart attack.

I should like to comment upon our approach to the heart. My associates and I exposed the heart at the operating table. We made direct observations of the organ and noted its response to experimental conditions. I should like to emphasize this point because the internist obtains information by use of the stethoscope, the X-ray, the fluoroscope, and the electrocardiogram. These are indirect methods of study. The surgeon can add direct vision to these indirect methods. It need scarcely be stated that direct vision gives a more accurate and complete picture than can be obtained by indirect methods.

The first point that I wish to make concerns the nature of the heart attack. The fatal heart attack is similar to a convulsive seizure. It is now recognized that an area of anoxemia in the brain gives rise to a convulsion. This focus in the brain has been referred to as a trigger. The trigger discharges and from it emanate impulses to other parts of the brain. These produce a generalized convulsion. An analogous condition exists in the heart. A trigger can be made by ligation of four or five coronary channels supplying an area of heart muscle two to three centimeters in diameter. This area of myocardium becomes cyanotic after these arteries are ligated and suddenly something happens which destroys the coordinated heart beat. Some fibers are contracted while others are elongated and the ventricles become a writhing, squirming mass of muscle which does not pump out any blood. The term ventricular fibrillation is applied to this condition, and as you know, it is fatal. It is similar to a convulsion of skeletal muscle and the term convulsion of the heart might well be applied to this condition. I believe if we could actually observe the heart in fatal heart attacks we would prefer to use the term convulsion of the heart rather than ventricular fibrillation.

I should like to return to the experiment in which we ligated arterial channels to produce a trigger. I should like you to visualize an experiment in which we ligate four small channels to a small area of myocardium. We can observe the cyanosis of this small area of myocardium. Now if we go no farther with our ligation we can close the operative wound and the animal will recover. If we go ahead and ligate the fifth channel to this small area of myocardium the trigger will discharge, destroy the co-ordinated beat and produce death. This fifth small channel then can be regarded as "the last straw." It makes the difference between life and death. This vessel carries a small amount of blood and a small amount of blood can make the difference between life and death. This small amount of blood can be looked upon as a blood-bath. I do not think we can over-emphasize the importance of a small amount of oxygenated blood delivered to that part of the heart where the blood supply is deficient. Those who are adversely critical of this research will please bear in mind that a small amount of oxygenated blood can be significant. It can save the life of the individual who has a trigger zone developing in the heart. I need scarcely remind you that when death occurs from a heart attack it is usually sudden and unexpected. Usually there are no signs of heart failure. This is a physiological derangement and it is as effective as turning the ignition switch in a smoothly working motor. The trigger zone may not be important anatomically. No doubt it could be excised experimentally if one cared to do so and the animal would be left with a good heart. A new set of coronary arteries is not necessary to prevent a small localized trigger from discharging. A blood-bath can be effective.

We next turn our discussion to the production of the blood-bath. How can this be done? There are three ways in which this can be done.

1. The first method is by the production of extracoronary communications. These are communications between the coronary arteries and the arteries of tissues grafted upon the heart. Various tissues can be used for this purpose. I used parietal pericardium, mediastinal fat, chest wall, omentum, spleen and other structures. Extracoronary communications were demonstrated by colored lantern slides. The coronary arteries of these specimens were injected with a mix-

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*From the Department of Surgery, The Western Reserve University and the University Hospitals, Cleveland, Ohio. This work was done before Lt. Colonel Beck entered the Army of the United States.

ture of barium sulfate and gelatin. The specimens were then cleared by the Spalteholtz method. The reader can see these specimens by turning to the November 1943 issue of the Annals of Surgery.

2. The second method by which a blood-bath can be produced is by the development of inflammation on the surface of the heart. The coronary arteries are end-arteries under normal conditions or almost end-arteries. Not only is each major coronary artery an end-artery but the secondary and tertiary branches of these arteries are end-arteries. This anatomical condition readily permits the formation of a trigger. Specimens of normal hearts were demonstrated.

Inflammation increases vascularity. When applied to the surface of the heart the vascularity of the heart is increased. Coronary arteries can be made to communicate one with another by producing inflammation on the heart. We found that powdered asbestos produced the most favorable response on the heart. Two-tenths of a gram applied to the surface of the heart produced granulation tissue of a highly vascular nature.

To measure the effectiveness of this procedure we ligated the descending ramus of the left coronary artery at its origin in one step in a group of normal hearts and in a group in which 0.2 gram of asbestos had been applied to the hearts at a previous operation. The mortality was 70 per cent in the first group and 31 per cent in the second group. There was a reduction in the size of the infarct in the second group. There was also evidence in the injected specimens to show that the asbestos produced communications between one coronary artery and another. Specimens were demonstrated.

These experiments are significant in that they do show that inflammation on the surface of the heart helps to prevent the formation of a trigger zone in the heart. These experiments clearly indicate this interpretation.

3. The third method by which a blood-bath can be provided by operation is to provide time for the occlusive disease to bring about intercoronary channels. In terms of the experiment if a coronary artery is occluded to such an extent that the heart beat is not destroyed and the animal continues to live then in the course of months, communication with the other coronary arteries develop. This is a very effective stimulus. It involves two components; one is arterial occlusion, the other is the factor of time. In the patient the disease process produces the occlusion. The factor of time is provided by extracoronary channels and by intercoronary channels produced by inflammation. Specimens were demonstrated showing communications between the coronary arteries. These channels are of good size. It is obvious that considerable protection against the formation of a trigger exists in specimens such as I demonstrated.

The patient who makes a recovery following coronary artery occlusion is in a position to develop intercoronary channels. It is reasonable to assume that the type of recovery varies with the development of these channels. The time factor is important. The longer the patient lives after the occlusion occurs the more favorable becomes the circulation. This statement assumes that the occlusive disease becomes static after occlusion occurred.

This presentation so far is based entirely upon the direct approach to the heart. I do not believe you will find it difficult to accept the concepts that are based upon this approach. The concept of the trigger mechanism is sound. The importance of a blood-bath is obvious and the three methods for the production of a blood-bath were demonstrated by injected specimens. This then constitutes the surgical approach to the treatment of coronary artery disease. The first difficult step has been taken. Another difficult step remains to be taken. The second step concerns application to patients.

Thirty patients were operated upon between the years 1935-1937. This small group of patients was regarded as a study-group. The results in the patients who survived the operation were good. Many of them were able to return to work and were relieved of pain. Seven additional patients were operated upon in the summer of 1942 before I entered the service.

Analysis of these results is not within the scope of this paper. But I would like to make some comment upon the subject of application. We know that a new set of coronary arteries cannot be given to the heart. We also know that a cicatrix in the heart cannot be replaced by good myocardium. These degenerative changes are irreversible. We also know that the greater the destruction the less effective is the pump. A degree of myocardial destruction can be reached when heart failure appears and a patient in failure is certainly no candidate for operation. Even if such a patient were to survive the operation, as was the case with one in this series, a good result cannot be expected. The heart when it is extensively scarred does not possess the property of recovery.

The desirable patient for operation is the type with little myocardial destruction. The operation should be looked upon as a prophylaxis rather than as a cure for damage once inflicted.

When occlusion occurs it is often too late to think of operation. The operation is not a drastic procedure. I think it could be done under local anesthesia.

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The precordial cartileges are taken out. The pericardium is opened and sutured to the precordium so that the heart comes into contact with the chest wall. The surface of the pericardium is roughened by means of a metal bur and a small quantity of asbestos is applied to the roughened surface. Powdered asbestos 0.2 grams is placed on the heart and along the internal mammary vessels. Mediastinal fat is brought into contact with the heart through openings made in the parietal pericardium. The wound is closed by sutures.

In the post-operative period a low grade inflammation occurs on the surface of the heart, on the surface of the parietal pericardium on the fat brought in from the mediastinum and along the internal mammary and intercostal arteries of the chest wall. It should be noted that these structures show increased vascularity. Not infrequently the patient feels better at the end of a week. This improvement in symptoms, no doubt, is due to the inflammation and the development of intercoronary channels.

The results obtained in the group of 37 patients operated upon for coronary artery disease appears in the November 1943 issue of the Annals of Surgery by Dr. Harold Feil.

TREATMENT OF BURNS

By FRASER B. GURD, M. D.
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There is an old cliché which refers to the impropriety of bringing coals to Newcastle. A similar remark might be made with reference to the presentation of a contribution upon the treatment of burns in Chicago since this city is the home of Sumner Koch and of others who have added much to our knowledge of the subject.

Early in the present phase of the War it became clear that the treatment of burns would become more important than during the first, and this not only among members of the armed forces but among civilians. As the result of aerial bombardment of civilians, fires in the cockpit and fuselage of aircraft, particularly of flash burns on shipboard and as the result of the employment by all combatants of tanks, an enormous number of burns have been treated. Those surgeons working in the battle areas and in Britain have asked for help from surgeons and physiologists on this continent; in consequence, moreover, of the large number of burns made available for study as well as treatment at Pearl Harbor, by the catastrophe of the Cocoanut Grove disaster in Boston and the almost equally catastrophic fire at St. Johns, Newfoundland, an opportunity has been given to many on this continent to observe methods for the handling of burned victims in large numbers.

Irrespective of the depth of the burn, it is unlikely that sufficient plasma loss will occur if less than ten per cent of the body surface be involved. When more than fifteen per cent of the surface, particularly if such involve the face and hands, the necessity for watching the patient for the development of burn shock becomes imperative. As plasma is lost from the blood stream into the damaged area haemoconcentration develops. This may be watched for by either the haematocrit or the haemoglobin and red cell methods of estimation.

As the percentage surface of the body damaged increases the degree of haemoconcentration also increases and the necessity for the administration of larger and larger quantities of plasma or serum becomes imperative.

In all larger burns it has been deemed necessary to administer very large quantities of serum or plasma if the patient is not to die within the first 24 hours; it may be, however, that it is possible to supply the necessary protein by means of oral administration at least after the first 36 hour period has passed. A recent experience in our clinic suggests that this may be so.

The contributions of Davidson in which tannic acid was advocated stimulated an enormously increased interest in the subject and during the following few years the death rate in most large clinics dropped. A further drop in the mortality rate in extensive burns occurred following the recognition of the nature of that type of shock which is responsible for the early death of so many victims. The establishment of blood banks and the consequent availability of plasma in relatively large quantities has resulted in the survival of many patients who previously died during the first 36-48 hours following injury. A third period of lessened mortality rate coincides with and is probably due in part at least to the introduction of sulfonamide therapy.

At the present time there exist proponents and protagonists of several essentially different methods for the local treatment of burns. These are (1) The employment of eschar producing techniques with the use of tannic acid either alone or with the employment of silver nitrate as an adjuvant, and of triple dye used either as jelly or solution. (2) The use of sodium chloride solution—or hypochloride—as bath, compress or bag (Bunyan). (3) The method which has been introduced by Blair, Brown and McDowell and developed by Koch and his associates and by Owen among others, and which has been made the basis upon which the technique employed at The Montreal General Hospital has been constructed.

It is not my intention this evening to attempt in any way a comparative analysis or critique of different methods for the local treatment of burns. I am going to presume that the reason why the management of this great organization has asked me to discuss the subject has been in order that I might place before you the technique which we have employed with considerable success at The Montreal General Hospital during the past two years, and which was described in November of last year in the Annals of Surgery. As we have already said, it has not been our intention to add to the confusion which envelops the subject of burn treatment but in a modest fashion to outline a method which, in our hands, has proven itself to be satisfactory, simple and safe.

We believe that it is necessary to have a plan for the treatment of major as well as minor burns. The plan which we have adopted at The Montreal General Hospital is based upon timing and, in order that this may be carried out, we have prepared a table with which I am sure many of you are familiar. For this purpose we have prepared a chart which is posted in all those parts of the hospital to which burns may be admitted. Such a chart makes it clear to everyone on the staff what should be done to commence the treatment of the burned patient immediately following admission and, we believe, makes it possible to carry on from one phase to another, with as little disturbance of the victim as is feasible, the various stages of therapy as they are indicated and shown on the chart. In this way the periods of shock and toxaemia, the possible period of sepsis and that of healing with or without skin grafting are presented in such a way that those in charge of the case always have the information required before them.

We have entitled this chart (which appears on pages 144 and 145)—

TIMING IN THE INFREQUENT OCCLUSIVE PRESSURE DRESSING TREATMENT OF MAJOR BURNS

It may be said that the superficial or second degree burn should heal promptly, that is within 12-14 days, no matter what form of local treatment is employed granted that the wound is not damaged by either destructive chemical or infection during this time. The question, however, is an entirely different one when so great destruction of the skin has occurred that skin grafting must necessarily be carried out. Three major surgical principles must form the basis of local therapy.

1. Prevent or at least minimize fluid loss.
2. Prevent infection.
3. Prevent contractures.

It will be noted in the following outline of the step-by-step procedure that is recommended that the first of these *desiderata* is accomplished by means of bulky compression dressings; that the second hoped for by adequate lavage and débridement, and the local application of sulfathiazole in an oil in water emulsion; and that early skin grafting or coverage is designed to prevent contractures.

The following description of the procedure employed in the treatment of major burns has been previously described in the November 1942 number of the Annals of Surgery by Ackman, Gerrie, Pritchard and myself.

It has been found best to have separate burn and shock teams, each composed of one attending surgeon, one resident and internes, all operating under a senior surgeon. Trained graduate nurses and orderlies are also part of our set up. Equipment includes special burn tents. Supplies should include an adequate amount of stored plasma or serum, 5 per cent sulfathiazole emulsion, and 4-inch by 6-inch cuts of 1 or 2 mm. curtain-mesh (Nottingham lace—11 point). Plumber's waste and 6-inch bandages are also necessary.

On admission, the major burns are placed at once in the burn tent, and the temperature gradually, not rapidly, raised to 80° F. The shock team functions first, estimating the haemoconcentration, and immediately administering plasma diluted 50 per cent by saline. This is done by a continuous drip in a cut-down intravenous, via the long saphenous vein at the ankle. A cannula, and not a needle, is used as a routine. It may be necessary to milk the plasma into the vein at the start. We have evolved a special double roller for this purpose.

When the above measures have been instituted, usually about one hour after admission, local treatment is begun. Operating room technique is used in the ward, in the tent bed, and only very rarely is anaesthesia employed. Morphine gr. one-quarter is administered on admission and repeated during the first hour if necessary (synergistic effect). For large surface areas (trunk or thigh) when life-saving is involved, tannic acid-silver nitrate eschar is permissible although we have given up all tanning methods. So far, we have not found a better type of eschar. In all critical areas, in moderate areas otherwise, and for all minor burns, we are using a thick pressure-type dressing. A mask is used for the face. We have modified the dressing by using a basic dressing of 5 per cent sulfathiazole oil in water emulsion (M.G.H. formula), as described in detail by Ackman and Wilson in 1942, and this has been found to be a highly successful method of preventing infection, or controlling it in delayed, contaminated, or actually infected cases. The details of the development and preparation of the emulsion, as well as our experience with it in burn therapy, and other surgical practice, have been described in the literature.

Local tissue concentrations of sulfathiazole when the emulsion is used are surprisingly high, 120 mg. plus per cent in 24 hours; blood levels are low and fleeting (maximum 3.5 in one hour) even with large amounts (10 ounces) of the emulsion. We recognize, as have others, the anaesthetic action of the sulfonamide, akin to the effect of benzocaine.

We find the use of the emulsion facilitates the softening and separation of slough in severe cases. It is not necessary in these cases to completely replace and renew the basic "sulfamesh" dressing. Generally speaking we find it quite sufficient to supply additional emulsion on the renewed gauze layer.

The M. G. H. emulsion, we believe, has distinct advantages over any other medium hitherto presented. It is simple and clean to handle. It offers economy in nursing and medical attention, and in the materials required. Its constituents are readily available and inexpensive. Sterility tests are negative, while its stability at extremes of temperature has been proven. The anaesthetic effect is noteworthy.

EMULSION SULFATHIAZOLE 5 PER CENT
(M. G. H. FORMULA)

Sulfathiazole (finely powdered)	5 per cent
Triethanolamine	2 per cent
Distilled water	24 per cent
White beeswax	5 per cent
Liquid paraffin	64 per cent

TECHNIQUE
(Step-by-Step Procedure)

1. Treat shock first.
2. With aseptic technique, cleanse burned area with soapsuds made from castille soap — without anaesthetic.
3. Wash with saline.
4. Apply single layer of 4-inch by 6-inch "sulfamesh" strips (1-2 mm. mesh), prepared beforehand or at time of treatment. Individualize fingers, etc. (This basic dressing may be left on at redressings.) Mask is used for the face.
5. Apply over this ordinary gauze dressings of 3-4 thicknesses, generously impregnated with emulsion. Do not individualize, fingers, etc.
6. Pressure dressing, with cotton waste outside this. Do not individualize fingers, etc.
7. Wrap in sterile towel or towels.
8. Bandage firmly, with 4-6-inch flannel or flannelette bandages cut on the bias, or dressing gauze roller bandages 4-6 layers in thickness, or ordinary roller bandage if the former are not available.
9. A plaster moulded splint is added for hand, forearm, etc., in the functional position.

Steps 6, 7, 8 and 9 minimize contamination. Note: In redressing, at whatever date, the "sulfamesh" is not changed unless débridement is indicated. This technique reduces pain and bleeding to a minimum, and permits removal of outer dressings to be performed more easily.

Although a few important surgical clinics, more especially that of the Massachusetts General Hospital, have recently indicated that cleansing of the wound by the employment of copious washing with soap and water and the removal of part at least of the devitalized tissue is not necessary, we are of the opinion that there is so little disadvantage in this procedure that, unless the emergency of the situation be such that too much time would be consumed by such a technique, it should be carried out.

In the case of burns of the second degree, that is those in which blebs have been present in the first instance, the dressing is not changed until such time, 10-14 days, that it is expected that the wounds will be healed. In the case of deeper burns in which it is thought that the removal of slough will be necessary, the dressings may be carried out after 6-8 days. The waste and dressing gauze are removed at first without disturbing the mesh. If on examining the limb it is clear that it is impossible to remove slough at this time, the mesh is not removed but further application of the emulsion, gauze, and waste is carried out.

Since it would appear that the protective property of the emulsion is largely lost after from 6-8 days, it is recommended that a change of dressings be carried out in third degree burns at such intervals.

It has been our experience that it is usually possible to remove the slough at about the end of 16 days so that soon after the completion of the third week skin grafting can be carried out.

As the result of the survival of a very much larger proportion of severe burns—20 per cent of the body surface, or over—new problems, more especially two, have become of preeminent importance. (1) Interest, and improvement, in the technique of covering burned areas in which destruction of skin has occurred. (2) The care of the victim who has gradually ceased to live in consequence of a nitrogen imbalance.

It is not unreasonable to affirm that during the past year or two an entirely new type of disease has become increasingly important in many clinics. This is exemplified by the patient who has suffered a burn involving 30 per cent, or more, of the body surface who in consequence of the exhibition of relatively large quantities of serum or plasma and of transfusions of whole blood has survived the first 48 hour shock period, has passed through the period of toxaemia, and whose wounds have remained relatively sterile so that death from sepsis has not supervened but who after two to five weeks lingering illness has gradually passed away. The clinical features of such a case are extreme emaciation with consequent asthenia and mental torpor, anaemia and marked hypoproteinaemia. In such a case large quantities of whole blood and plasma have been introduced intra-

TIMING IN THE INFREQUENT OCCLUSIVE PRESSURE

		PERIOD OF SHOCK 48 HOURS	
	First Hour	**First 24 Hours**	**Second 24 Hours**
General Treatment	Hypo Morphine ¼ gr. (Intravenous) if necessary Repeat in the hour if necessary	Repeat Morphine as may be indicated during this period	
Shock Team 24 hour service		Cortin 50 cc. (optional) in extreme cases	Repeat Cortin Intramuscularly — 20 cc. - 30 cc. Q. 6 H. as indicated for 48 hours
Under Supervision of Senior Surgeon	**Intravenous Therapy** Plasma or Serum In amounts of 500 cc. First 500 cc. given rapidly	Continue to a minimum of 1,000 cc. (Plasma or Serum) for 10% burn in 48 hours by continuous intravenous drip. Control the amount of serum or plasma by Haemoglobin, Haematocrit and Plasma Proteins—as well as clinical observation	
	10% Glucose only if indicated	Intravenous 10% Glucose as indicated, add saline if vomiting	
	External Heat—Develop slowly to 70° F. under tent	SERA A.-T.-S. — 3000 units Anti-Gas Serum when indicated	
	Water by mouth containing carbohydrate for first 24 hours		Fluids—As tolerated high in Carbohydrate
	Oxygen by B. L. B. apparatus or tent — when indicated		Continue Oxygen as indicated
			Enema
Local Treatment	Delayed until Intravenous Shock Treatment begun and effective	**Operation** Without anaesthetic using pure soap lather (detergent when necessary)	
Burn Team		Mechanical removal of debris, dirt and dead tissue	
		Open blebs	No dressing change
Under Supervision of Senior Surgeon		**All Burns** **Occlusive pressure dressing** over 5% Sulfathiazole Emulsion (Ann. Surg., Nov., 1942—Vol. 116, — page 641)	
Laboratory Examinations	Haemoglobin Estimation or Haematocrit and Plasma Proteins R. B. C. Count	Repeat Q. 1 H. until Haemoconcentration controlled and thereafter at least Q. 4 H.	Continue Haematocrit, Hgb. and Plasma Proteins Q. 4 H.
		Culture of burned surface before treatment	**Blood Chemistry** Plasma Proteins Chlorides Sugar Sulfonamide Level
	Urinalysis	Urinalysis	Urinalysis
Clinical Data	Admission T.-P.-R. and Blood Pressure	Chart P. & B. P. Q. 1 Hr. Haematocrit, Plasma Proteins, Haemoglobin %, Temperature } Q. 4 H.	T.-P.-R. Q. 4 H. B. P. and P. Chart Q. 1 H. Haematocrit, Haemoglobin and Plasma Proteins
Chart		Intake and Output Daily Plasma. Serum. Glucose. Saline. Blood Chemistry; Sera	Intake and output daily amounts of all types of infusions separately and in units

DRESSING TREATMENT OF MAJOR BURNS

TOXEMIA PERIOD	GRANULATION PERIOD	HEALING PERIOD
2nd to 5th Day	**6th to 14th day**	**2nd to 6th Week**
Morphine or Barbiturates as indicated	Codeine or Barbiturates when indicated	Codeine or Barbiturates when indicated
Sulfonamides orally or intravenously not desirable especially if urine volume is below 1,000 cc. per day		Sulfonamides only indicated orally if pneumonia threatens
Blood Substitutes as indicated	**Fresh Blood Transfusions** at least 500 cc. Q. 3 days, and depending on R. B. C.	**Fresh Blood Transfusions** at least 500 cc. Q. 3 days, and depending on R. B. C.
Glucose as indicated, by blood chemistry **Saline** only when blood chlorides depleted	Infusions if indicated	
Fluids—Minimum of 3000 cc. by mouth	Fluids—3000 cc. daily	Fluids—2000 cc. daily
Diet—High in Protein and Carbohydrate	**Diet**—Controlled by Blood Chemistry	**Diet**—Same Tube Feeding if necessary for Hypoprotenaemia
	Vitamins and Iron	Vitamins and Iron
Enema—Q. 2 days P. R. N.	Cathartics as indicated	Cathartics as indicated
	Superficial Burns (1st and 2nd°) Remove dressing on 8th to 10th day because healed as a rule	**Superficial Burns**—Usually Healed. No Dressing
	Deep Burns (3rd°) Re-dress and remove debris on the 12th - 14th day	**All Deep Burns**—Change every 12 to 14 days
No dressing change	**Anaesthetic if necessary**	
	Graft as soon as possible	**Grafting at each change**
	Homografts if necessary	
	Occlusive pressure dressings in all cases, including donor areas	**Occlusive pressure dressings in all cases, including donor areas**
Continue Haematocrit, Hgb. and Plasma Proteins daily as indicated Blood count daily	Continue Haematocrit, Hgb. and Plasma Proteins daily as indicated Blood count daily	Haemoglobin and Red Blood Count and Plasma Proteins at least twice weekly
Blood Chemistry—Sugar, Urea and Creatinine if indicated by Toxaemia or Urinary Suppression Sulfonamide Level if necessary	**Blood Chemistry**	**Blood Chemistry**—as indicated Sulfonamide level if necessary 3 days
	Culture with dressings	Culture with dressings
Urinalysis daily	Urinalysis Q. 2 days	Urinalysis
T.-P.-R. Q. 4 H. B. P. as indicated Haematocrit, Haemoglobin, Plasma Proteins	T.-P.-R. Q. 4 H. B. P. as indicated Haematocrit, Haemoglobin, Plasma Blood Counts Proteins daily	T.-P.-R. Q. 4 H. B. P. as indicated Haematocrit, Haemoglobin, Plasma Blood Counts at least twice weekly Proteins daily
Intake and Output Plasma and all other infusions, sulfonamides and progress notes	Same daily including blood transfusions Notes on granulation and operations	Notes on granulation and operations

venously during the past two or three years but almost invariably the result has been that gradually the patient has deteriorated and died.

With the advent of this new clinical picture the result, as indicated, of the employment of serum and plasma during the early hours, several observers, more especially Robert Elman of St. Louis, and Charles Lund and his associates at the Boston City Hospital, have apparently been successful in pointing the way for a correction of the nitrogen imbalance which is clearly responsible for the exitus of these cases.

This is not the proper place nor is time available to discuss in any detail the experimental work nor the clinical observations which have been responsible for the improvement in therapy in this connection. The September number (1943) of the Annals of Surgery is largely made up of contributions dealing with this subject, more especially those of Lund and Elman referred to above. During the past nine months at The Montreal General Hospital as the result more especially of personal communications from Lund, we have employed the technique which has been developed by him in burned patients and in others suffering from marked hypoproteinaemia. In this connection we have been impressed by the prompt response to the introduction into the gastro-intestinal tract of the high protein feedings as recommended by Lund. In a recent case of 50 per cent burn the method of high protein introduction into the gastro-intestinal tract would appear to indicate that this method may be used to conserve plasma.

EMPYEMA

By JOHN ALEXANDER, M. D.
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Empyema is too prevalent a disease to be as badly treated as it is in many places. Essentially, empyema is an abscess in the pleural cavity. As the pus increases, the lung is pushed farther and farther from the chest wall. In the early days of a typical empyema the pus collects in the bottom of the pleural cavity because the pus gravitates there by virtue of the patient's sitting in bed or lying in bed with one or two pillows under his shoulders.

The most important thing I wish to bring out at the beginning of this talk is that in the early days of the illness, the empyema cavity, the abscess, is not an encapsulated one, not limited by adhesions; in the course of time pleural adhesions form at the limits of the pus collection, localizing it.

The chief cause of empyema is pulmonary infection; some type of infection of the lung almost always precedes the development of the pleural infection.

Surgical drainage should be carried out only when the following conditions are fulfilled or, more importantly, drainage must not be used unless the following conditions are fulfilled: First, drain only after pyogenic organisms have been found by smear or in cultures of the aspirated pus. This provision is made because some patients unexpectedly will be found to have a pure tuberculous empyema even though they may have the characteristic appearance of having a non-tuberculous empyema. Second, the empyema cavity must first be reduced to a small size by aspirations every one or two days. If open tube drainage is immediately produced there is a fair chance that the patient will die from collapse of the lung and pendulum respiratory movements of the mediastinum because adhesions have not yet limited the collection of pus and fixed the lung to the chest wall. The physiologic effect is that of a sucking wound of the chest wall.

Why should the abscess be reduced by repeated aspirations? In the first place, to overcome the mediastinal displacement if the empyema is large enough to produce mediastinal displacement. In the second place, progressively to reduce the size of the empyema cavity and to cause adhesions around a smaller and smaller pleural abscess. In the third place, progressively to reduce the toxic absorption from the pleural abscess.

The third condition that must exist before drainage is that the aspirated pus be no longer thin. It was discovered by the Empyema Commission of the Army in the last war that when the aspirated pus contained from 75 to 90 per cent sediment, sufficiently firm pleural adhesions had formed to prevent collapse of the lung at the time a tube drain was put in place. If one drains before the pus has become from 75 to 90 per cent sediment, the lung is apt to collapse and convert a partial empyema into a total one of a whole hemithoracic cavity. Determination of the amount of sediment is made by keeping a test tube of aspirated pus upright for twelve hours, when layers of sediment and supernatant fluid will be clearly differentiated.

Graphically one sees in this slide a total empyema. After aspiration of, let us say, 1,000 cc. of pus on the first day and 500 or 800 cc. on the second day, and probably in decreasing amounts every one or two days, the empyema will, after one or two weeks, have been reduced to a relatively small localized pocket. Then, when the pus probably contains from 75 to 90 per cent sediment, one knows that pleural adhesions have formed and that when a tube is introduced the lung will not collapse and allow infection to occupy the entire pleural cavity.

When the patient is able, he should spend most of the time in the semi-Fowler position so that the lung will progressively expand from before backward and limit the final collection of pus that is to be drained to the posteroinferior portion of the pleural cavity.

It is the practice of some physicians to inject air at the time of aspiration or to allow some air to leak into the pleural cavity at the time the syringe is disconnected from the needle in order to be emptied. The presence of air above the pus in an empyema cavity creates a serious complication. One should never permit air to enter the pleural cavity in the management of a non-tuberculous empyema. As you know, one often substitutes air for the pus of a pure tuberculous empyema in order immediately to establish an artificial pneumothorax for the treatment of the tuberculosis that we know exists in the lung at the time a pleural empyema forms. When air is injected or allowed to enter during the aspiration, this air floats to the top of the pleural cavity and permits the lung to collapse at the apex of the pleural cavity. This combats the very object one is attempting to attain, namely, the production of gradual expansion of the lung. This slide illustrates the persisting collapse of the upper lung by the air that has been admitted to the pleural cavity, in spite of the gradual expansion of the lower lung following dependent surgical drainage. Obliteration of such a superior empyema pocket is difficult to accomplish and requires the passage of a long tube from below upward to the remaining empyema cavity at the top of the hemithorax.

(Slide) Here is an illustrative case. This patient had had an empyema for some weeks before this x-ray was made. Extensive fibrinous pleural adhesions existed over the lower part of the pleural cavity. Air had presumably been allowed to enter the chest at the time of the initial aspiration, and the air floated to the top. Then, when drainage was established, the last part of the lung to expand would be the apical portion; this long narrow drainage track between the expanded lower lung and the ribs needed to be maintained in order effectively to drain the apical portion of the empyema, which never would have existed if air had not been allowed to enter. Many months were required for the complete obliteration of this empyema cavity, whereas it probably would have become healed in a few weeks if air had not been allowed to enter the pleural cavity.

There is always danger of air's entering the pleural cavity if one uses only a needle and a syringe and if more than one syringeful of pus is to be aspirated, because when one disconnects the needle from the syringe in order to empty the syringe, air is apt to rush into the needle and then into the chest. The simplest of all ways to prevent this is to have between the syringe tip and the needle a short length of rubber tubing that may be pinched or clamped with a hemostat while the syringe is being emptied. If, however, one is going to aspirate a large amount of pus it is easier to use a three-way stopcock, which permits one to aspirate any number of syringefuls of pus, and discharge them into a basin, without once disconnecting the apparatus. Air, therefore, cannot enter the pleural cavity.

I have spoken of when one should, and when one should not, drain in typical cases of empyema. In typical cases drainage is always deferred until repeated aspirations have greatly reduced the size of the empyema and caused its limitation by adhesions. But in certain atypical cases one must drain immediately, under the following circumstances: When the aspirated pus has a foul odor, indicating a putrid empyema. If one does not drain immediately, a spreading cellulitis or phlegmon of the chest wall is likely to develop because of the deposition of highly virulent anaerobic organisms in the needle track after withdrawal of the needle. Such a cellulitis is obviously a grave complication. We now have a rule that if, on aspiration, the pus is found to be foul the patient should be taken immediately to the operation room and an incision made through the needle track so as to prevent a spreading cellulitis.

Also, one drains immediately if a pulmonary abscess has suddenly ruptured into the pleural cavity, producing an acute empyema; if an empyema is complicated by a large bronchopleural fistula; if the pus is so thick that one cannot satisfactorily aspirate it through a large needle; and if there is a recurrent or chronic empyema, with or without a pleurocutaneous sinus. If there is such a sinus, one should attempt to reintroduce a drainage tube of adequate size. If the previous drainage wound has closed completely, one must reoperate and introduce a sufficiently large tube to establish proper dependent drainage.

(Slide) This is an illustration of the thoracic wall of a patient who had a putrid empyema which was aspirated and not immediately drained. Cellulitis spread rapidly in the chest wall and required very wide resection of the necrotic tissue, together with chemotherapy and x-ray therapy in order to save the patient's life.

Where does one drain an empyema in order to drain it dependently? One of the greatest mistakes in the drainage of empyema is drainage above the floor of the empyema cavity. There is no rule of thumb as to the particular rib one should resect, because the organizing fibrin that sediments down in these empyema cavities causes adhesion of the diaphragm to the thoracic wall at varying levels above the anatomic floor of the pleural cavity. The simplest way to determine the position of the floor of a particular empyema cavity is to aspirate from the normal lowest limit of the pleural cavity upward until pus is encountered; this represents the lowest limit of the particular empyema cavity. One chooses the posterior axillary line in empyemas that are in the posterolateral lower chest (as most are) because this position furnishes effective dependent drainage. Although drainage in the scapular line would be equally dependent, patients will not lie on a freshly introduced rubber tube that is in this position; they will turn on their sides and then drainage is not dependent. If the tube is placed dependently in the posterior axillary line so that the mattress will not press against it, the patient will lie upon his back and enjoy dependent drainage.

In acute empyemas, however, the diaphragm is likely to rise after drainage for the distance of a rib and an intercostal space. Drainage will, therefore, soon be dependent if the tube is introduced through the bed of the resected rib above, rather than below, the lowermost intercostal space from which pus was aspirated. If the tube is placed lower, the elevation of the diaphragm may interfere with the tube, requiring that it be introduced farther in order to reach the empyema cavity.

With regard to the localization of the floor of atypically located and shaped empyemas, lateral projection x-rays are often necessary. In this slide of a posteroanterior projection x-ray, we see a basilar empyema but we do not see this second empyema pocket that we clearly see in this lateral projection x-ray in the mid posterior chest entirely separated from the empyema seen in the posteroanterior x-ray. In many cases, at least those that are not typical, one must have a lateral projection x-ray in order adequately to localize the empyemas for dependent drainage.

Until the last few years we used to believe that we could not produce air-tight drainage unless we pushed a catheter in between the ribs through a canula introduced through a short incision in the skin. All of us have used this method of drainage and I suppose none of us has failed to be discouraged at the way such catheters usually behave. In the first place, the trocar and canula introduction of a catheter is dangerous if one undertakes to introduce it dependently, because the trocar may wound the lung that may be adherent to the diaphragm, or may perforate the diaphragm and seriously injure either the liver, spleen, colon or stomach.

After one has obtained localization of an empyema by waiting for the production of pleural adhesions while daily aspirations of pus are carried out, one does not risk the sudden collapse of the lung if he opens the pleural cavity for a few moments before he introduces the drainage tube. The only efficient way to maintain prolonged air-tight drainage is to introduce a large drainage tube after a short segment of a rib has been resected. So, after the pus has become from 75 to 90 per cent sediment as the result of repeated aspirations every one or two days, one makes a short incision, usually vertical, through the skin and extracostal muscles and resects five or six centimeters of a rib at the floor of the empyema cavity, or just above it, as determined by intercostal aspirations. An incision is then made in the deep layer of the periosteum and the underlying parietal pleura that is not a bit longer than enough to permit the snug introduction of a thumb-size drainage tube. Such a tube, introduced in this way, will stay air-tight for months, even though one does not suture the extra-costal muscles, which he may do, but he should not suture the skin because of the danger of infection.

The drainage tube is, of course, not left open. Its outer end is put under water in a bottle on the floor. Because the bore of the tube is so great, pus will flow through it freely. Had the tube been a catheter, clots of fibrin and pus would soon occlude it and then the pus would force its way out around the tube and air would begin to suck in around the tube; usually in two, three or four days the air-tightness of catheter drainage is lost, but when the incision in the deep layer of the periosteum of a resected rib and the underlying parietal pleura is only long enough to permit the snug introduction of a thumb-size tube, drainage will remain air-tight indefinitely.

This is a photograph to illustrate the type of drainage we use routinely. In the first place, this air-tight drainage is clean. The dressings do not become grossly soiled. This vertical incision is five or six centimeters long. The safety pin that is passed through the side wall of the tube is held in place by two strips of adhesive plaster that are crossed over the ends of the safety pin and not merely across the bar; this method of holding the pin will keep the tube firmly in position for a long time. The tube is long enough to permit the patient to turn freely in bed. When the patient inspires, the water from the bottle rises in the tube for several inches, indicating the amount of negative pressure in the pleural cavity. On expiration, the water drops, but not to the level of the water in the bottle. Continuous negative pressure is thus maintained in the drainage tube and, therefore, in the empyema cavity, constantly pulling on the lung and causing it gradually to expand and obliterate the empyema cavity. Only by the expansion of the collapsed lung does an empyema cavity become obliterated. To a lesser extent the diaphragm rises, the mediastinum shifts and to a very small extent the ribs fall in, but obliteration of empyema cavities is brought about chiefly by expansion of the lung.

In the postoperative management after drainage, air-tight, water-sealed drainage is used until the cavity is less than 30 cc. in capacity. The capacity and the dimensions of the cavity should be measured weekly. One makes sure that the tube is always in proper position; that is, that it is not too far in the pleural cavity, and that it is in far enough. The tube should be shortened only when the cavity is completely obliterated. One of the greatest mistakes in the treatment of empyema is the removal of the drainage tube as soon as the discharge becomes scanty. If this is done, some patients will get well in spite of it, but in many cases the opening through the thoracic wall will heal rather rapidly and leave a mildly infected residual pleural space which in the course of days, weeks or months will again become actively infected, leading to a chronic empyema.

I just spoke of frequently measuring the capacity and dimensions of the empyema cavity so that one may know exactly where he stands in the management of a particular patient. There are four methods of measuring a cavity. One of the simplest and best is palpation of the limits of the empyema cavity with a uterine sound which may be curved to the desired degree; this is done after the temporary removal of the drainage tube, which should be cleaned before being reintroduced.

By elevating the lower chest or the hips with a pillow or pillows, one may introduce a measured amount of fluid in order to learn the capacity of the cavity, provided, of course, that there is no bronchopleural fistula.

A method for measuring a small residual empyema cavity is palpation of it with a gloved finger after temporary removal of the tube. When the track is a long and perhaps sinuous one that does not permit the ready introduction of a uterine sound, one elevates the hips and instills lipiodol through a catheter introduced to the top of the cavity and then takes an x-ray while the patient is still on his side.

The causes of chronicity of empyema teach us in retrospect what to avoid in the treatment of acute and subacute empyema. The most important of all the causes is mismanagement of drainage. Too early drainage is bad because it allows the whole lung to collapse, substituting a total hemithoracic empyema for one that perhaps was a relatively small one before the tube was introduced. Too late drainage means that scar tissue has already formed on the visceral pleura and will tend to bind down the lung and prevent its ready expansion. Drainage of too short duration permits the chest wall drainage track that was created surgically to close, thereby sealing an infected pleural cavity that is not filled by lung. Too narrow a tube, such as a catheter, has already been mentioned; it does not efficiently drain off the fibrin and pus clots. Too short a tube permits the parietal pleural layer to granulate over the end of the tube and block drainage. Too long a tube acts as a standpipe and pus only drains out when it reaches the high end of the tube. Drainage that is not dependent leaves a dependent pocket of pus that maintains infection in the walls of the empyema cavity. Undrained pockets obviously leave one or more empyemas untreated. Drainage that is not air-tight fails to subject the lung to the constant suction of the negative pressure that air-tight water-sealed drainage creates.

Other causes of chronicity which in the aggregate are important but which all together are not as important as the failures properly to conduct tube drainage, are rather obvious: persisting pyogenic, tuberculous or actinomycotic infection in the walls of the cavity; excessive scar of the lung or visceral pleura; bronchopleural fistula; esophagopleural fistula; communication with infected structures such as a pulmonary abscess or bronchiectases draining into the empyema cavity; subphrenic or perinephritic abscess; necrotic neoplasm of any thoracic organ; foreign body in the pleural cavity, such as a lost drainage tube, a sequestrum from a rib, or a bullet or shell fragment.

If a patient with a chronic empyema is not being properly and dependently drained at the time you first see him you should create dependent adequate drainage and should take a biopsy of the parietal pleura in order to determine whether tuberculosis exists as a possible cause of chronicity. The drainage should, if possible, be made air-tight and water-sealed. If the cavity is closing too slowly after a trial of one or two weeks of such drainage, some form of mechanical suction drainage should be used. If these various things fail in the course of several months, one is forced to carry out a deforming combined extrapleural and Schede thoracoplasty.

This slide illustrates a case in which dependent suction drainage of four months completely obliterated an eight months' empyema that had never been adequately drained. As a result of the contraction of the scar tissue in the chest wall in this chronic empyema, a scoliosis had occurred; as a result of breathing exercises the spine straightened.

This slide illustrates the type of suction drainage I use. It is very simple. This tube comes from the patient and enters an empty bottle (which serves merely as a pus trap) through an air-tight lid. This bottle is connected by means of a rubber tube with a tall bottle containing water. Another tube is connected with this bottle and leads to some form of mechanical suction. Both tubes enter the tall bottle through an air-tight lid but neither tube is submerged beneath the water in the bottle. A glass tube, with its outer end open passes through the lid of the bottle, the inner end of the tube being submerged in the water. The depth to which the tube is submerged in the water determines the amount of negative pressure in the drainage system and, so, in the empyema cavity. As soon as the mechanical suction becomes greater than the negative pressure desired, air is sucked down the glass tube and bubbles up through the water to enter the air space above the water. If we start with 15 cm. of negative pressure and the patient is comfortable with this amount, we add, two or three days later, another 2 or 3 or 5 cm. of water in the bottle, thereby submerging the glass tube farther and lowering the pressure in the chest farther. Davison's mercury suction apparatus creates greater negative pressure than even a very tall water-filled bottle.

This slide shows the hugely thickened, fibrotic parietal pleural scar that occurs in chronic empyema and that is one of the reasons why certain neglected empyemas cannot be cured in any other way than by a deforming resection of the ribs, the intercostal bundles and this greatly thickened unyielding parietal pleural scar. The extent of the deformity resulting from a Schede thoracoplasty obviously depends upon the extensiveness of the resection of the thoracic wall which, in turn, depends upon the size of the chronic empyema. The seriousness of chronic empyema is the chief reason why advantage should be taken of every means at our disposal to treat acute empyema effectively so that the development of a chronic empyema will be prevented.

Some Facts about the Sex Steroid Family

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Hormones, even at present, are regarded by many clinicians as vague ephemeral substances. Their humoral ancestry and the past impotency of their therapeutic products, to a large degree, are responsible for this viewpoint. It is our purpose to review certain facts about one of these hormone families. It is our hope that this review will reveal the sex steroids as sturdy pharmacologic individuals which are capable of accomplishing good and doing harm when applied to therapy.

Endocrine Families

The hormones fall into two chemical families. A knowledge of the characteristic traits of these families facilitates their application to therapy. The hormone families are:

1. The steroid family, and
2. The amino or protein family.

The *steroid family* includes the active principles of the gonads and the adrenal cortex. Important members of this family are:

1. Ovarian estrogens—estradiol, estrone and estriol;
2. Corpus luteum hormone—progestin;
3. Male hormones or androgens—testosterone;
4. Adrenal cortical steroids—including desoxycorticosterone; and
5. Various intermediate or metabolic products of these steroids.

Like Rabbit in Milne's Winnie the Pooh the steroids have many "friends and relations." Among these friends and relations, which are not hormones, are the synthetic estrogens. These include diethylstilbestrol, hexestrol, and triphenylchlorethylene. There are numerous other synthetic estrogens.

The *amino or protein family* contains all the remainder of the hormones. Some of these are amines or amino acids. Others are peptids or proteins. Among the members of this family are:

1. Thyroid protein—thyroglobulin—comprised of the iodine containing amino acids, diiodotyrosine and thyroxin;
2. Insulin, a protein;
3. Epinephrine, an amine;
4. The Gonadotropins—pituitary, chorionic and equine—which are glycoproteins; and
5. The active principles of the posterior and anterior pituitary and the parathyroids which are protein-like.

The steroid and amino or protein families are distinguished by different general characteristics.

The *steroids* are higher alcohols and have chemical properties of alcohols. They are well known chemically. Their structural formulas have been derived. They are available as pure crystalline substances. They are stable, do not require refrigeration and commercial preparations of these carry no expiration dates. They have no allergic propensities since they are non-protein. The steroids are soluble in vegetables oils which are their best solvents for intramuscular administration. They are active by mouth to various degrees. Estrone sulfate is the most active. Diethylstilbestrol is one of the most orally active of the synthetic estrogens. The steroids are active locally when applied to skin or mucous membrane. Dosage of these steroids may be expressed in terms of milligrams.

The *amino or protein hormones* have the properties of amines, amino acids or proteins. They are poorly known chemically except epinephrine and thyroglobulin. With the exception of insulin, epinephrine and thyroxin, none is available as a pure crystalline compound. The majority, therefore, exists in rather impure extracts. With the exception of insulin and thyroxin, none of these hormones is stable. The majority requires refrigeration and deteriorates with age. Allergy is not uncommon. The amino or protein hormones are commonly dispensed in aqueous solutions, save for thyroglobulin. Only thyroid protein is active by mouth. The remainder of these hormones is destroyed by peptic digestion. Save for thyroid protein, the amino or protein hormones are active by hypodermic and some by local application to the mucous membrane. Dosages of these hormones must be expressed in biologic units except those of thyroid substance, thyroxin and epinephrine.

CHEMISTRY OF THE STEROIDS

All of the steroids are characterized by a common characteristic configuration. The basis of their chemical structure is the phenanthrenecyclopentane nucleus. This nucleus relates the steroids to cholesterol, stigmasterol, certain digitalis glucosides, and bile acids, various opium alkaloids and some of the bufotoxins. These substances are distant cousins of the sex steroids. Cholesterol, however, or some closely allied steroid, may be the matriarch of the entire "phenanthrenecyclopentane clan."

The adrenal cortex appears to be a veritable brooder of steroid compounds. More than twenty-five of these steroids have been isolated from the cortex. Ten of the cortical steroids are pharmacologically active. These are corticosterone, 11-dehydrocorticosterone, 17-hydroxycorticosterone, 11-dehydro-17-hydroxycorticosterone, 11-desoxycorticosterone, 11-desoxy-17-hydroxycorticosterone, progesterone, 17-hydroxy-progesterone, adrenosterone and estrone. The first seven of these which include progesterone possess cortin-like properties. Progesterone, of course, also has progestational properties. 17-hydroxy-progesterone and adrenosterone are weakly androgenic. Estrone naturally is estrogenic. It is doubtful that any of these steroids represents the true cortical hormone.

Only one crystalline hormone of high potency has been identified in the testis. Six androgenic steroids have been isolated, however, from normal tissues and body fluids. These are testosterone (from beef testes), adrenosterone (from beef adrenals), androsterone (from male and female urine), isoandrosterone (from male and female urine), dehydroisoandrosterone (from male and female urine) and epi-allo-pregnanol-3-one-20 (from pregnancy urine).

Testosterone is seven to ten times more biologically active than androsterone. Dehydroisoandrosterone is 40 per cent as active as androsterone. Isoandrosterone is 10 to 15 per cent as active as androsterone.

The ovaries and the chorio-placental system secrete two groups of sex steroids, estrogens and progestin. Two estrogens, estradiol and estrone, have been identified chemically in sow ovaries. The estrogens, estradiol, estrone and estriol, have been isolated in crystalline form from the placenta and from the urine of pregnancy. Estriol has been found only in human tissues and fluids. Estriol is present in the urine as an ester of glucuronic acid, estriol glucuronide. Estrone probably occurs in the urine as a sulfuric acid ester; estrone sulfate has been recovered from the urine of mares.

Progesterone is the only progestin which has been recovered from the ovaries of slaughter house animals. The chemical nature of the corpus luteum hormone of the human ovary has not been identified. No appreciable quantities of progestational steroids are present in the urine. A biologically inert steroid, pregnanediol, is thought to be one of the major urinary excretion forms of progestin.

The estrogens differ chemically from the other sex steroids by the fact that they do not contain an extra methyl group. Androgens, progesterone and the cortical steroids contain this group.

METABOLIC OR EXTRAGENITAL PROPERTIES OF STEROIDS

The physiologic and pharmacologic properties of steroids are of two general types:

1. Metabolic or extragenital, and
2. Genital.

The genital properties are emphasized more commonly than the metabolic ones. Some of the metabolic properties are reviewed.

1. *Steroids and Water Electrolyte Metabolism.* All of the sex steroids favor the retention of water and sodium. The order of their activity is desoxycorticosterone > testosterone > progesterone > estradiol. Desoxycorticosterone increases potassium excretion. Testosterone increases potassium retention. Estradiol apparently does not alter potassium excretion.

2. *Steroids and Protein Metabolism.* The cortin-like steroids deplete body protein for glyconeogenesis and, thereby, nitrogen elimination is favored. Testosterone and estradiol favor the storage of body proteins and, thereby, body protein is stored. The steroids tend to increase muscle efficiency through their effects on water and sodium metabolism. Androgens and estrogens increase the volume and dynamic strength of skeletal, cardiac and smooth muscles. Testosterone is more active in this respect than estradiol. The myotropic action of estrogens chiefly is upon uterine, ureteral and bladder muscles.

3. *Steroids and Carbohydrate Metabolism.* The cortin-like steroids favor glycogen storage and glyconeogenesis. Desoxycorticosterone, the most commonly employed of the cortical steroids, has little effect upon the carbohydrate metabolism. Testosterone, estrogens and progesterone have no significant role in carbohydrate metabolism.

4. *Steroids and Osseous System.* Testosterone and estrogens favor lineal bone growth and osseous ma-

turation. Estrogens enhance the deposition of calcium in the organic matrix of bone.

5. *Steroids and Vascular System.* The cortin-like steroids inhibit vasodilatation. Testosterone, estrogens and progesterone favor vasodilatation. Cortin-like steroids and testosterone increase blood flow by their direct action on cardiac muscle.

6. *Steroids and Urinary System.* The steroids favor the resorption of water and sodium by the renal tubules. Their order of activity in this regard is cortin-like steroids > testosterone > progesterone > estradiol. Testosterone and estrogens increase renal and bladder volumes. Progesterone relaxes bladder and ureteral muscles.

7. *Steroids and Skin.* The steroids favor hydration of the skin. Testosterone and estrogens increase skin pigmentation. The hyperpigmentary state of pregnancy is due to an estrogen plethora. Testosterone and estrogens are responsible for the characteristic growth of genital crines.

Genital Properties of Steroids

The adrenal cortex must be added to the ovary and testis in listing the sex glands. Some of the reasons for this are:

1. The cortex produces androgens, estrogens and progesterone.

2. Castrates of both sexes excrete in the urine androgens, estrogens and pregnanediol (a metabolic product of progesterone). The obvious inference is that these substances are of cortical origin.

3. Virilizing and feminizing syndromes arise from cortical hyperplasia and cortical tumors.

The hormonal pattern of sex is not as simple as it was formerly thought. Males and females excrete the same androgens in their urines and in the same quantities (androsterone, isoandrosterone and dehydroisoandrosterone). Both males and females excrete estrogens in their urines. Estradiol, estrone and estriol have been found in female urine. So far only estrone has been found in male urine. A sexual differential arises, however, because the adult female is characterized by estrogen levels which are approximately three times those of the adult male.

1. *Source and Import of Androgens in the Female.* The androgens in woman are doubtlessly of cortical origin. The urinary values for androgens are not significantly reduced by castration and may even increase following castration. The urinary androgens increase during the climacteric.

The androgens doubtlessly have significant metabolic or extragenital roles in woman. No genital roles of androgens in woman have been established. We conclude, therefore, that the gynecic functions occur despite woman's androgenic moiety and not because of it.

2. *Source and Import of Estrogens in the Male.* Experimental studies in laboratory animals supply data that androgens are formed by ovaries and that estrogens are formed by testes. It is well known that stallion urine is one of the richest sources of estrogens and that stallion testes contain more estrogens per gram than any organ of pregnant or non-pregnant woman. Nevertheless, estrogens in man are probably adrenal in origin. An important reason for this belief is that urinary estrogens do not disappear from the urine when a male is castrated. The fact that estrogen levels are more impaired in non-surgical hypogonadism than androgen levels suggest that the site of production of these two groups of steroids is not the same. Estrogens have, doubtlessly, significant metabolic or extragenital roles in man. No genital roles for estrogens in man have been established. We conclude, therefore, that andrologic functions occur despite man's estrogenic moiety and not because of it.

3. *Physiologic Genital Effects of Estrogens in Woman.* These are well known. Estrogens produce sexual differentiation and maturation and maintain the functional integrity of woman's generative system. The principal targets of the estrogen growth stimulus are the vulva, vagina, cervix, endometrium, myometrium, tubes and breasts.

4. *Physiologic Genital Effects of Progesterone in Woman.* These are well known. Progesterone is concerned with prenidatory preparations and with the protection, growth, nutrition and the sheltering of the embryo. The primary targets of progesterone activity are the cervix, endometrium, myometrium and breasts.

5. *Contraphysiologic Effects of Androgen Therapy of Woman.* Since no physiological genital roles have been established for androgens in woman, it may be inferred that androgen therapy of woman is without factual foundation. When androgens are given, woman is defeminized and masculinized. Targets of this androgenic action include the skin (hirsutism), larynx (husky voice), clitoris (enlarged), vulva, vagina, cervix and uterus (atrophy), and ovaries (suppression of function).

6. *Physiologic Genital Effects of Androgens of Man.* These are well known. Androgens produce sexual differentiation and maturation and maintain the functional integrity of the generative tract. The principal targets of the androgen growth stimulus are the penis, scrotum, epididymides, seminal vesicles, prostate and Cowper's glands.

7. *Contraphysiologic Effects of Estrogen Therapy of Man.* Since estrogens have been accorded no physiologic genital role in man, estrogenic therapy of man has no foundation save for patients in whom depression of testicular function is advantageous. Estrogens when administered to man produce demasculinization and feminization. Targets of this contraphysiologic estrogen action include: the penis and scrotum (decreased in size); testes and epididymides (volumetric decreases, depression of spermatogenesis and degeneration of seminal epithelium); prostate and seminal vesicles (decrease in size and reduction in secretion—decrease in the volume of seminal plasma) and the breasts (gynecomastia).

SUMMARY

The steroid family possesses common family traits. These are manifest in their chemistry and in their physiologic and pharmacologic properties. The steroids have important metabolic or extragenital roles. At times these roles of individual steroids may be similar or dissimilar. Androgens are the monitors of the andrologic system of the male and estrogens are the monitors of the gynecic system of the female. When gonadal failure results in deficiencies in the gonadal steroids, therapy with androgens in the male and with estrogens in the female yields satisfactory results, if the fact is borne in mind that this therapy is substitutional, not curative, not stimulational and does not circumvent the associated sterility. The cheapness of estrogens has made substitutional therapy in woman more practical than substitutional therapy with androgens in man. Since no occasion arises in clinical practice for virilizing women, androgen therapy of woman is without rationale. Carcinoma of the prostate has afforded an indication for contraphysiologic treatment of man with estrogens. This therapy is rational. By demasculinizing man, depressing testicular function and thereby androgenic levels, the growth of prostatic malignancy is reduced. Furthermore, the specific role of estrogens in calcium metabolism—the laying down of calcium in the matrix of bone—has an important role in the healing of osseous metastases of prostatic carcinoma.

The pharmacologic properties of the sex steroids should be familiar to anyone employing them in therapy. Metabolic actions may result in untoward responses even though the elected action is soundly indicated. All use of the sex steroids should be upon rational grounds. Hormonal empiricism with the impotent organotherapeutic substances of the past was relatively safe: the patient usually was not harmed or helped. The empiric use of the potent steroids, which are available now, may do great harm.

REFERENCES: No attempt is made to cite the numerous studies of various workers which have afforded this synthesis of present concepts. A number of these is given in the following publications of the author: *Endocrine Gynecology*, Charles C. Thomas, Springfield, Ill., 1939; The Sex Steroid Family, *Journal-Lancet* 61:393, 1941; and *Endocrinology of Woman*, Charles C. Thomas, Springfield, Ill., 1944.

Tumors of the Spinal Cord

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Fifty-five years have passed since the first successful surgical removal of a benign spinal cord tumor by Victor Horsley, after it had been diagnosed and localized by Gowers. Since that time there has been a gradual and extensive accumulation of data regarding the nature, behavior, and treatment of tumors involving the spinal cord. From a clinical standpoint, any new contributions to the subject can represent little more than refinements of our knowledge.

These tumors are not common, and yet they deserve our attention because they are frequently misdiagnosed and because approximately 65 per cent of them are amenable to successful surgical treatment. Furthermore, an error in diagnosis may lead to useless abdominal or pelvic operations, various types of injections, and possibly dangerous spinal manipulations. Almost without exception patients who harbor spinal cord tumors have been treated for numerous non-existent or irrelevant disorders before the correct diagnosis is made. The delay incident to such errors increases the duration of spinal cord compression, with possible irreversible damage to nervous tissue.

When confronted by the problem of pain in the back, the extremities, or the trunk, it is quite natural and usually correct for the physician to consider the likelihood of the more common conditions, such as orthopedic disorders, or disease of the thoracic or abdominal viscera. The possibility of a spinal cord tumor, however, should be borne in mind if serious diagnostic errors are to be avoided.

Tumors may occur within the spinal canal at any level from the foramen magnum down to the sacrum, and they may occur at any position in the transverse relationship of the spinal canal. For example, they may occur in the vertebral body, or in the extradural space as in the case of certain types of sarcomas and occasionally extradural meningiomas. The most common site, perhaps, is the intradural space outside the spinal cord. In other words, the commonest types of tumor are located outside of the cord but within the dura. Occasionally one may arise from the vertebral arch, and rarely a chondroma may arise from the intervertebral disc.

The types of tumors which are most common are those which lie in the extramedullary intradural space, namely, tumors of the meninges and tumors arising from the sheaths of the spinal nerve roots. Meningioma is the term given to tumors which arise from the arachnoid or from the inner surface of the dura. There is considerable controversy among pathologists about the origin of these tumors, but the concensus of opinion is that they arise from the arachnoid, although at operation most of them seem to be intimately attached to the dura. The neurofibromata arise from the nerve sheaths of the spinal nerve roots. These two tumors account for 65 per cent of primary tumors involving the spinal canal and its contents.

Outside of the dura, the most common are the sarcomas. Within the substance of the cord various types of glioma are found. These are not as common as those in the extramedullary location.

According to Elsberg, 56 per cent of spinal tumors occur in the thoracic spine, inasmuch as it is longer than the other parts. The majority of them are extramedullary, and the smallest number arise within the substance of the cord itself.

From a clinical standpoint, the most important tracts that are affected in the process of spinal cord compression are the pyramidal tracts, which control the motion of the arms and legs, the spino-thalmic tract carrying the sensory fibers of pain and temperature in the anterolateral portion of the cord, and sensory tracts in the posterior aspect of the cord which carry the sensory impulses of touch and vibration and two-point discrimination.

Neurofibromata arise from either the anterior or the posterior nerve roots. If they arise from a posterior nerve root, they usually give rise to pain or some sensory change.

The neurofibromata and the meningiomata are slow in growth. The development of these tumors often covers a period of several years. As they increase in size, they gradually compress the cord on one side or another and produce symptoms referable to various tracts, particularly the main sensory tracts and the pyramidal tracts.

The history in these cases varies somewhat, but the commonest initial symptom is pain. That pain is often localized in some portion of the spine, or it may radiate along a nerve root and be referred to the arms or legs, or thorax or abdomen. Pains of this nature simulate such disorders as angina pectoris, biliary colic, ureteral stone, kidney stone, intercostal neuralgia, various pelvic disorders, so-called sciatica and disorders of the various joints, so that one must be on guard continually when attempting to determine the cause of pain.

As the tumor develops, compression of the cord occurs with resultant disorders in motor power and in sensation, until in the later stages of compression a paraplegia with paralysis of the legs or arms, or of all four extremities may ensue, depending upon the level of the cord involved. If it is in the cervical region, all four extremities may be involved, whereas if it is in the thoracic region or lower, the legs alone will be involved.

The sensory changes may be gradual, beginning with parasthesia on one side or the other of the body, and gradually increasing until both sides of the body are involved. In the typical final stage of cord compression it is often possible to identify a "sensory level" below which there is anesthesia or hypesthesia.

Various sphincter disturbances may also be present in cases of spinal cord tumor. Patients must be questioned regarding disorders in control of the bladder and rectum.

In the general physical examination of these patients it is important to consider the possibility of a primary tumor in some other part of the body, for metastases to the spine are not uncommon from tumors of the breast and prostate, from hypernephroma and from other viscera. These must be borne in mind, particularly in older patients. Neurofibromatosis or von Recklinghausen's disease of the skin may be associated with tumors within the spinal canal, usually neurofibromata attached to the nerve roots. Blood counts should also be checked for possible blood dyscrasias, because certain types of leukemia and lymphoid diseases invade the spinal canal and in some instances the cord itself and may produce symptoms of spinal cord tumor. The blood Wassermann is of importance in every case because of the frequent necessity of differentiating syphilis of the cord and spinal cord tumor.

It goes without saying that the neurologic examination is the most important part of the entire examination of a patient who is suspected of harboring one of these tumors. The motor function of the extremities must be checked carefully; alterations in muscle tone, hyperactive or diminished tendon reflexes, changes in the abdominal or anal reflexes, and pathologic signs, such as the Babinski sign or ankle clonus, aid in localizing these growths. The sensory innervation of the skin is arranged in zones or dermatomes corresponding to the segments of the cord from which the nerves originate. It is easy perhaps to remember that the fourth thoracic dermatome passes through the nipple line and the tenth thoracic dermatome passes through the umbilicus. The lower sacral nerves supply the so-called "saddle area" around the anus and the buttocks, a consideration which is of particular importance in connection with tumors of the cauda equina and of the lower end of the cord, the conus medullaris.

Patients who harbor tumors in the lumbar part of the spine, particularly in the case of large tumors such as the ependymoma, usually have pain for many months or years localized in the lower spine and may be treated for arthritis and many other orthopedic conditions before the possibility of tumor is suspected.

The x-ray is of considerable help to us in some instances. Anteroposterior, lateral, and occasionally oblique views of the spine are taken. A good film which shows clear details of the pedicles and intervertebral foramina as well as the vertebral bodies is to be desired. Erosion of the pedicles and widening of the spinal canal, when present, usually indicate the presence of a tumor within the spinal canal at that level.

At this point it is essential to emphasize the most important aid in the diagnosis of spinal cord tumor. The history, the physical examination, and neurologic examination, and the x-ray of the spine are all important, but whenever a spinal cord tumor is suspected, the major diagnostic aid is lumbar puncture. Many laymen and not a few physicians still hold the impression that lumbar puncture is a major operation, but when properly employed by one who is skilled in its use, it is a simple and harmless procedure, which is essential not only in the diagnosis of tumors of the spinal cord but also in many other neurologic diseases. When a needle is placed in the lumbar subarachnoid space and a glass manometer is attached to it, the cerebrospinal fluid rises in the glass manometer to a certain level. In the normal case the fluid column pulsates with the arterial pulsations and with the respiratory pulsations. In the presence of a block in the spinal canal from tumor, the arterial pulsations are feeble or absent entirely. In the absence of spinal subarachnoid block, compression of the jugular veins of the neck, as described by Queckenstedt, increases venous pressure within the cranium, and this increased pressure is transmitted to the cerebrospinal fluid causing a prompt rise of the fluid level in the manometer. At least 90 per cent of cases of spinal cord tumor show a complete or partial block in the spinal canal by the Queckenstedt test. It is true that in a few cases a tumor may be present without evidence of block. In some cases the spinal fluid may rise promptly on jugular compression and fall very slowly on release of compression. The fluid is often yellow below a spinal cord tumor, and there is almost always a definite increase in the protein content of the fluid. Quantitative determination of the total protein should be made in every case.

In the presence of a large tumor of the cauda equina, such as ependymoma of the filum terminale, which may fill the entire lumbar portion of the spinal canal, lumbar puncture frequently fails to obtain fluid. In such a case the puncture may be unusually painful due to the fact that the nerve strands of the cauda are held in a fixed position by the tumor and therefore cannot be easily pushed aside by the advancing needle point.

Occasionally neurologic examination fails to localize a tumor with sufficient accuracy to determine the proper site for laminectomy, and it is helpful to resort to lipiodol studies of the spinal canal. Two cc. of heavy lipiodol is injected into the cisterna magna through a spinal needle inserted between the atlas and the foramen magnum, and the lipiodol is allowed to run down the spinal canal while the patient is lying on a fluoroscopic tilt table with the head elevated. The descent of the lipiodol is retarded or completely blocked at the level of the tumor. This level indicates the site for operative exposure. Usually the neurologic examination is sufficiently accurate in localization to permit a laminectomy without using this procedure.

In the differential diagnosis of spinal cord tumors it is possible to mention only briefly some of the conditions which must be considered. Syphilis of the cord, various types of myelitis, circumscribed arachnoiditis which may produce a block by the Queckenstedt test without any increase of protein in the fluid, and ruptured intervertebral disc which may simulate a tumor of the cauda equina must be borne in mind. Primary tumors of the vertebrae, such as various types of bone tumor, must be considered. Degenerative diseases of the cord, such as multiple sclerosis, amyotrophic lateral sclerosis, syringomyelia, and combined sclerosis of pernicious anemia must be considered in differential diagnosis.

When the diagnosis of spinal cord tumor has been made, laminectomy should be performed and the tumor removed if possible. Almost all meningiomata and neurofibromata can be completely removed, with very little danger of recurrence except in the rare cases of malignant variants or metaplasias of such tumors. After relief of cord compression by such tumors, the outlook for return of normal neurologic function is very good, particularly if the compression has not lasted for more than two years. In glioma of the cord or extradural sarcoma the prognosis is definitely poor because of the infiltrative or malignant character of the growth; roentgen therapy may be employed with temporary or palliative benefit in such cases. Laminectomy carries with it an almost negligible risk to the life of the patient.

Finally, a plea should be made once more for the consideration of spinal cord tumor in the differential diagnosis of painful conditions or disorders characterized by motor, sensory, or sphincteric changes referable to disease of the cord. Prompt diagnosis may mean the difference between permanent invalidism or death and restoration to normal function.

DIAGNOSTIC DERMATOLOGICAL CLINIC

The Diagnosis and Treatment of Commoner Skin Conditions

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I have chosen to discuss this morning the subject of the eczematous dermatoses because I know of no more important subject in dermatology to the man in general practice than the subject of eczema and dermatitis.

The name "eczematous dermatoses" is given this group because there is still a great diversity of opinion as to what eczema is. Walker called eczema "a term commonly applied to any wet or scaly inflammation of the skin of the cause or nature of which the observer is ignorant."

There is one group of dermatologists who take the position that eczema is a disease due to some endogenous causation, at least it is never exogenous, while on the other hand there is another group which feels that eczema has nothing distinctive about it, that it is nothing more than dermatitis. Sutton says "eczema has a usage analogous to that of rheumatism; it may mean anything. All cases of eczema are to the pathologist dermatitis. Eczema implies chronicity, a tendency to recurrence, an element of idiosyncrasy, and the absence of complete etiological analysis."

Out of this apparent confusion we are able to differentiate several different types which we believe will somewhat clarify the situation, and it is of these that I wish to speak this morning.

The first is contact eczematous dermatosis. This is an inflammation of the skin due to occasional continuous or intermittent contact with a cutaneous irritant. These irritants consist of a wide variety of substances, among the most common of which are plants, chemicals, dyes, soaps, drugs, and various substances encountered in the home or in industry. The dermatitis may be acute or chronic. It is characterized as acute by redness, edema and the formation of papules and vesicles. It may produce simply scaling or desquamation or it may progress to oozing, weeping and crust formation. Pruritus is nearly always a distressing symptom and the ensuing scratching often leads to secondary pus infection. In other cases, due to continued exposure a simple dermatitis will become chronic, infiltrated and lichenified. Clinically we may find any one or a combination of these lesions present.

This type of dermatitis may be caused by any number of irritants. The concentration of the irritant, the length of contact and the conditions under which the exposure occurs differentiate the types of dermatitis into non-sensitization and sensitization dermatitis. Non-sensitization dermatitis is caused by an irritant that in given concentrations and under given surrounding factors affects nearly all human skins. An example of such an irritant is a powerful chemical applied deliberately or accidentally to the skin. Sensitization dermatitis, known as contact eczematous dermatosis, on the other hand is a dermatitis due to repeated exposures to substances normally innocuous. The initial exposure may produce no visible irritation, but subsequent exposures may produce a dermatitis at the local site or a generalized reaction.

Among the common causes of sensitization dermatitis are plants such as the primrose, chrysanthemum, tulip, daffodil, narcissus, the Rhus family of poison ivy, poison oak, sumac, and the ragweed group. Other common irritants are hair dyes, clothing dyes, depilatories, nail polishes, cosmetics, adhesive plaster, dress shields, novocain, and so forth.

Patients with thin, sensitive skins are especially predisposed to contact eczematous dermatosis. In some individuals an allergic change is responsible for this predisposition. There is a distinct susceptibility on the part of some people to irritation, while in others this predisposition is due to scratching, friction, sweating, maturation and traumatism of the affected parts.

While in the beginning a patient may be sensitive only to one specific allergen or irritant, this sensitivity may in some cases be followed by a sensitivity to many substances, a so-called poly-sensitivity.

In making a diagnosis one must take a careful, detailed history, noting especially the patient's occupation, his habits, and the location of the eruption. When obtaining a history of exposure one must note the time that has elapsed since the last exposure because the resulting eruption generally develops from twelve hours to several days after exposure.

In the case of Rhus poisoning, however, the incubation period may be even longer. Inasmuch as the eczematous reaction is due to external irritation, the exposed areas are the most frequent sites for the development of the dermatitis. For example, in dermatitis due to cosmetics, the eyelids and sides of the face and neck are the locations affected; in occupational dermatitis the dorsa of the fingers and hands; in dress dye and dress shield dermatitis, the axillae; in poison ivy, the legs, hands, forearms and genitalia; in hatband dermatitis, the forehead; in dermatitis due to white gold spectacles, the area behind the ears.

One might think that chronic dermatitis on the hands of housewives would always point to laundry soap as the allergic agent, but in most cases the etiologic irritant is discovered only by careful detective work in following up every clue. In many cases the definite localization of the eruption, the history of sudden onset, and careful questioning, will bring out a clear-cut history of exposure to an allergenic irritant.

In differentiating contact eczematoid dermatitis, we must consider three or four other types of eczema: first, nummular eczema; second, atopic eczema; and third, infectious eczematoid dermatitis.

I shall attempt to differentiate these conditions and show you, by means of slides, what we mean by the different types.

(Slide) This is a case of hair lotion dermatitis. This patient went into a barber shop for a scalp massage and the barber applied a lotion containing resorcin. You see the result. The patient was allergic to resorcin.

(Slide) This is a case of dermatitis due to the application of another scalp lotion, a preparation known as "Process-Kura." This old gentleman wanted to stimulate his scalp, and this is the result.

(Slide) This is a cold cream dermatitis. Very few cold creams produce dermatitis. I believe the offending agent in most cold creams is the perfume that is used, and some people are allergic to the perfume. In this case the irritant was a perfume known as methyl heptin carbonate.

Now, what shall we do for patients with these acute dermatoses, especially the one I showed first with the lesions on the face due to hair tonic? Of course, the first thing to do is to thoroughly remove the offending agent by washing the scalp with soap and water. The most useful agent in acute weeping dermatoses (and we must always think of soothing preparations in these cases) is aluminum subacetate. This is used as wet compresses, an ounce or two tablespoonsful to the pint of water. After two or three days of such treatment the swelling disappears, the patient feels a great deal more comfortable, and a mild ointment, such as a 2 per cent ichthyol ointment in equal parts of Lassar's paste and cold cream, or a mild boric acid ointment, may be employed. It should be borne in mind that soothing remedies are those indicated.

In this case we would employ such treatment, namely aluminum subacetate wet compresses, followed by a mild 2 per cent ichthyol ointment.

Where the eruption is not on the face and the patient does not mind a little staining, another valuable preparation is silver nitrate solution ¼ to ⅛ per cent as a wet dressing. Some localities are having difficulty in obtaining aluminum subacetate, and in those areas silver nitrate solution can be substituted.

(Slide) Nail polish dermatitis is a very common condition nowadays and is often overlooked. Not every person who wears nail polish develops dermatitis, but many do. The characteristic lesions are generally seen on the eyelids. A woman comes in complaining of itching of the eyelids and of an eczematous condition there. Again, she may have lesions on the side of the neck, as this girl has, on the eyelids, or on various portions of the face. The itching is sometimes intense. The skin is often dry, scaling, and sometimes lichenified.

(Slide) Another case showing the marked involvement of the eyelids. After the polish is removed the response is rather quick and the local treatment consists merely of using simple ointments, such as 2 per cent ichthyol in Lassar's paste and cold cream or ordinary zinc oxide ointment. The nail polish should, of course, be removed immediately.

(Slide) Another example showing the markings on the side of the neck due to nail polish. We see an average of three or four cases of this a week despite the fact that there has been considerable publicity given to the condition in the health columns of the newspapers. The exact ingredient in the nail polish responsible for the cutaneous irritation is not known.

(Slide) This is a severe dermatitis due to the use of an ordinary innocuous substance. It is on the market, extolled very highly as a soothing remedy after shaving and for sunburn. It didn't work out that way here. One would naturally ask this patient what she had been using. The treatment here would be aluminum subacetate wet dressings until the edema and most of the inflammation had subsided. After that a soothing cream should be employed.

(Slide) This is not a common condition but it is a lipstick dermatitis. Ordinarily lipstick doesn't cause much trouble, but occasionally we do see irritation from it. Removal of the lipstick, of course, and discontinuance of its use, followed by a soothing ointment is all that is necessary.

(Slide) This shows considerable dermatitis about the mouth, the chin and the eyelids. It turned out to be a case of dermatitis due to contact with soap and with a tooth paste. We occasionally see irritations about the lips and the mouth that are taken for vitamin deficiencies but are really due to the orris root in tooth paste. That contact should always be investigated as a possible cause of dermatitis about the mouth and lips.

(Slide) This is a case of Butesin Picrate dermatitis.

I will now show a few examples of self-medication dermatitis. This patient applied Butesin Picrate for a mild burn on the fingers. This ointment does not always react in this fashion but we see a great many cases where the patient is definitely allergic to Butesin Picrate. The treatment here, of course, is aluminum subacetate wet dressings or ¼ to ⅛ per cent silver nitrate wet dressings, followed by soothing ointments. In other words, the more acute the inflammation, the milder should be the treatment.

(Slide) This is a case of phenol-camphor dermatitis. You all know that when moisture is present the phenol in the mixture is liberated, producing a phenol burn. This is an especially bad location to use the preparation because the feet are generally moist in many patients with ringworm of the feet.

(Slide) This is a very severe dermatitis due to the use of black wash for an ordinary irritation of the hands. The patient was susceptible to mercury and this is the result. We treat this the same as the other cases, namely wet compresses followed by soothing preparations. Do not use strong preparations in a case like this.

(Slide) This is a case of contact dermatitis due to the application of Metaphen in a patient who was definitely allergic to Metaphen.

(Slide) This patient, poor soul, has a very severe dermatitis venenata or contact eczematoid dermatitis, due to the fact that she used some foot salve that had been given her son for ringworm of the feet. The salve was probably Whitfield's ointment and, as you can see, it served as a very marked irritant to the face. You might think the condition was erysipelas except for the absence of temperature, the absence of an advancing border, and the symmetry of the eruption. The treatment to pursue is the usual one of wet compresses of aluminum subacetate solution. Occasionally you will find a case that will be irritated by a dilution of one ounce of aluminum subacetate solution to a pint of water. In those cases we use an ounce to two pints of water or we use an ounce to a pint of aluminum subacetate and add to that a pint of boric acid solution. Lead acetate, provided it is well diluted, is another good preparation to use.

(Slide) This is a severe dermatitis in a patient who had psoriasis and who applied sulphur and lard. Sulphur is all right, but it is a dangerous drug to use unless you are sure of your ground. It can be safely used in scabies for three or four days, as you know, but it must not be used continuously because sulphur and tar will produce dermatitis in susceptible individuals so we must be very careful about its use in percentages stronger than 2 or 3 per cent except in the case of scabies. The old-fashioned remedy of equal parts of sulphur and lard was used here with this result.

Strange as it may seem, though, in some patches of localized chronic dermatitis we have been getting excellent results with 40 per cent sulphur, made up as a paste. It relieves itching and does not produce a great deal of irritation even in 40 per cent strength.

(Slide)' This is a dress dermatitis. In cases of dress dye dermatitis, the offending agent is a dyed dress, not necessarily a cheap dress, and the irritant is paraphenylendiamine in the dress material. Blue and black dresses are the commonest offenders. The dermatitis is seen on the neck and chest and on the exposed parts not covered by the underwear. It is also seen in the axillae. In the case of dermatitis in the axillae, though, we have to think of dress shield dermatitis because dress shields will often cause an eruption of this type.

(Slide) This is a case of "shorts" dermatitis. We are not seeing much of that now, but for a while there was almost an epidemic of "shorts" dermatitis in Chicago. The shorts were manufactured well but the trouble lay, as discovered by Dr. Louis Schwartz of the United States Public Health Service, in the resin that was used to finish the material. This resin was also the agent which was responsible for the dermatitis that gave Nylon stocking such a bad reputation at first. The area covered by the short is the area involved and the diagnosis can be made quite readily.

(Slide) Another case of "shorts" dermatitis, a very severe dermatitis.

(Slide) This is a case of adhesive plaster dermatitis, an occupational affair. This man was dressed for a wound on the third finger. After a few days the dressing was removed and a simple adhesive plaster applied with this result. He was in the hospital about three weeks recovering from a severe eczematoid contact dermatitis due to adhesive plaster.

(Slide) This is a cutting oil dermatitis. We are seeing a great deal of it now due to the use of so much cutting oil in the war industries. Cutting oil dermatitis is characterized by an acneiform and furuncular eruption involving generally the hands and the extensor aspects of the forearms.

(Slide) This is a case of dermatitis due to satinwood contact.

(Slide) This is a surgeon who developed a latex dermatitis from his rubber gloves.

(Slide) A case of dermatitis due to contact with benzine.

(Slide) Sodium cyanide dermatitis.

(Slide) Soap dermatitis.

(Slide) This man was a painter in constant contact with turpentine and the paint materials finally produced this dermatitis.

(Slide) A cleaner of upholstery in one of the hotels here.

(Slide) Adhesive plaster.

(Slide) Primrose dermatitis. This is a very common condition in the spring around Eastertime when old ladies are apt to receive primroses from their friends, also at Christmastime. It is an itching dermatitis confined to the forearms, hands, and sides of the face. It resembles poison ivy dermatitis somewhat.

(Slide) This is a toilet seat dermatitis due to contact with the lacquer on the toilet seat. This is easily diagnosed by noting the location of the eruption on the buttocks just where they come in contact with the toilet seat.

(Slide) This is a dermatitis from a leather watch strap. This is also easily diagnosed because of the location of the eruption.

(Slide) This illustrates the way patch tests are performed. This is a positive test with nail polish. A small amount of the material to be tested, be it face powder, nail polish, a piece of dress material, or shoe leather, is moistened and applied directly to a portion of unaffected skin. This is then covered with a larger piece of gutta percha or oiled silk, held in place with a larger patch of adhesive plaster, and left on the site for 24 to 48 hours. Patch testing, unless executed with caution, may entail dangers. If a substance is applied at the incorrect time, on the incorrect site, or in an incorrect manner, it may produce both local and generalized reactions which can incapacitate the patient for a long period of time. Many substances when applied in concentrations can actually produce new sensitizations. However, the use of certain forms of patch testing is relatively simple and safe.

Positive patch reactions may occur with substances which have nothing to do with the causation of the eruption so the reaction to tests must therefore be correlated with all other available evidence.

(Slide) This is a case of sulfathiazole dermatitis. I have not the time today to go into that subject but in the current literature there are numerous reports of sulfathiazole dermatitis. The cases that we have been seeing are those whose skin has been previously sensitized by the application of sulfathiazole to eczematous areas, followed by the internal administration of the drug.

(Slide) Leaving the subject of contact eczema, I would like to discuss briefly several other types of eczema.

This is a case of nummular eczema. Nummular eczema is a form of vesicular dermatitis that occurs in coin sized patches and affects primarily the dorsal surfaces of the hands and the forearms, and the extensor aspects of the legs and thighs.

It appears at any age, is always worse in the winter, clearing in the summer. It recurs frequently. It is seen in housewives, dish washers, and surgeons. Foci of infection in the teeth, tonsils, and prostate are predisposing factors while exposure to soap and water, acids, and alkalies favor its development as well as do exposure to oil and grease.

The eruption begins as pin head sized papules and vesicles which become grouped then confluent, forming patches which vary in size from a dime to a dollar. They are few or numerous, moist or oozing, and often crusted. Lesions often clear in the center, spread peripherally and show some resemblance to ringworm.

This type of eczema is a difficult one to treat. Since many cases improve in the summer, one should simulate summer conditions by generalized ultra violet lamp exposures and administration of vitamin A and D in large doses. Mild antiparasitic treatment is more efficient than soothing anti-eczematous measures.

Painting these lesions with 5 per cent crude coal tar in chloroform is of value, so are wet dressings of silver nitrate solutions, ¼ to ½ per cent, as well as wet dressings of potassium permanganate solution, Gr. V to the quart of water. When the lesions are dry, crude coal tar ointment is of great value.

The next three slides are those of nummular eczema.

The next group, and a most important one, are the atopic dermatoses.

(Slide) This is a baby with infantile eczema, the first step in the development of atopic eczema.

The atopic dermatoses are the group which is intimately associated with the atopic form of sensitivity. There is a close association between asthma, hay fever, migraine headaches, and certain dermatoses, such as eczema in infants, so-called prurigo, and disseminated neurodermatitis.

This slide is a typical case of infantile eczema, a type very common in babies. The essential cause is unknown; often there is a familial history of some form of atopy, either asthma, hay fever, migraine, or eczema. Most babies with eczema are fat and overfed. Eggs, milk, oatmeal, and meat juices are believed by some to be causal factors. Most cases are made worse by soap, by contact with dust, wool and silk, and by exposure to cold winds. The disease has a characteristic localization on the flush areas of the cheeks, the forehead, and the chin. It may remain limited there or extend to the scalp, wrists, legs, and trunk. The affected skin is red, rough, and fissured with minute cracks which ooze serum. The disease often responds quickly to treatment but recurrences are common. The disease often lasts through the first two years of life.

In treating such a case, the baby's skin should be cleansed with olive or mineral oil. Soap should never be used. The clothing should be light, soft, and cool. Contact with silk and wool should be avoided. House dust and the substances emanating from mattresses, pillows, and beds and rugs are often allergenic factors, and they should be avoided if possible. The child's diet should be carefully watched, and it is often advantageous to change the type of milk, substituting goat's milk, boiled milk, or evaporated milk for cow's milk. During the period of food testing, discontinue temporarily orange juice and cod liver oil. The child should be restrained from mutilating the skin by having him wear cardboard splints in the bend of the elbows, by pinning down the arms at night, and by the wearing of a soft muslin mask.

Locally tar affords more relief than any other ointment. Crude coal tar used in full strength or diluted with equal parts of vaseline or Lassar's paste often affords great relief.

(Slide) This slide shows a typical case of atopic dermatitis in a young adult. The areas affected are the face, sides of the neck, and upper chest, the cubital fossae, and the popliteal spaces. The affected areas are dry, infiltrated and lichenified. Itching is severe and crusted and secondarily infected areas are commonly seen.

Asthma is very often an accompanying symptom and juvenile cataracts have occurred.

No eczema is more difficult to treat. These patients suffer intolerably. They are always worse in cold weather, when they are tired, and when they are emotionally upset.

In treating this type of case, itching must be alleviated or the patient will mutilate the skin. The best topical remedies are crude coal tar ointment, ointments containing liquor carbonis detergens, aluminum subacetate, and phenol in ½ to 1 per cent strength. Soap should never be used for cleansing. As a substitute, sulphonated oils are useful and quite beneficial to the skin.

Dust, wool, silk, and occasionally rayon, through contact and inhalation, are irritants and should be avoided. Certain foods, chocolate, eggs, nuts, cheese and pork, are allergenic in many cases. A change of scene and a change of climate are most beneficial.

I am sorry that my time does not allow me to discuss infectious eczematoid dermatitis.

DIAGNOSTIC SURGICAL CLINIC

The Diagnosis of Low Back Pain

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The symptoms of backache with referred pain along the course of the sciatic nerve has for many years attracted the attention of orthopedic surgeons and industrial surgeons. In early days, all pains in the lower back resulting from injury were diagnosed as "railroad spine" and the patient was classed as a malingerer. Then came Goldthwaite, with his theory that this type of pain was all caused by sacroiliac subluxation. Practically every backache, whether or not accompanied by sciatic pain, was attributed to strain, to subluxation or to sacroiliac disease. This theory held for a number of years. Later, Theodore P. Willis showed that more than 7 per cent of 748 specimens as they passed through the laboratory showed variations in the lumbosacral region; that this region was the site of more irregularities, congenital anomalies and unusual anatomy than any other in the body; and that these variations could account for many of the symptoms and combinations of symptoms exhibited in the lower back and affecting the nerves emerging from that portion of the spine. His findings rationalized certain conclusions made previously on a clinical basis, regarding the cause of low back pain. In 1916, I stated before the American Roentgenological Society that more consideration should be given to the fact that the lower back is composed of fascia, muscles, joints with cartilage and synovia, tendinous attachments, interlacing ligaments, all surrounding a bony canal through which the peripheral nerves course and exist; and that any inflammatory reaction, whether caused by strain, infection, allergic reaction, gout or other forms of toxemia, can and does produce the same symptoms that more recently have been attributed to rupture of an intervertebral disk.

If there is any one place in the body where a fine differential diagnosis is necessary it is the lower back. Mechanically it is of weak structure for the work it is called upon to do. It is the fulcrum on which every motion of the body is pivoted, at the junction of a flexible and an inflexible part, the lumbar spine, the sacrum and the ilia. The tissues which compose the lower back are subject to the same ills as tissues elsewhere in the body, and mechanical strain exerted on these tissues is much greater than elsewhere. Added to this, in no other location are there as many anatomic variations in structure as can be found at the lumbosacral junction. All these factors contribute to making it an area where pain can occur with great suddenness and can be brought on by various causes.

In the very recent literature which I have searched thoroughly, the names of nerosurgeons stand out preeminently as contributors on the subject of low back pain accompanied by so-called sciatica. Their approach has been largely from the standpoint of pressure from a ruptured intervertebral disk as the major cause of such back pain. To one who has been interested in the subject for more than thirty years, and has examined many hundreds of such cases, this viewpoint is much too narrow. It is not the intention to contend that low pack pain is not or could not be caused in some cases by rupture of a disk with pressure on the nerves or their roots, but to point out that the same symptoms can be caused through disarrangement of other structures. It seems strange that Schmorl, in more than 7000 autopsies found only two cases of ruptured nucleus pulposus, whereas our own surgeons have operated on hundreds of cases in the last five years.

It is about thirty years since I performed my first bone graft for immobilization of the lower spine. For more than twelve years I have made sections of the joints from the lower spine of every case operated on, and in every recurrent chronic case of backache with sciatica pathology was found in the cartilage of the joints, with varying degrees of inflammatory reaction around the joints. It seems worth while to repeat that the tissues forming the lower back are subject to the same influence as like tissues in other parts of the body. The mechanical weaknesses of the lower back are known to all of us, and in my opinion it is a combination of mechanical weakness plus other contributing causes, which many times produces the same symptoms that particularly have been ascribed to ruptured intervertebral disk. To list these symptoms briefly is apropos at this point:

Pain in the back accompanied by sciatic pain.
Pain usually accentuated by coughing or sneezing or straining at stool, which temporarily increases intraspinal pressure.
Night pain, or pain occurring in the early hours of the morning, causing the patient to walk the floor or sit in a chair.
Paresthesia in the distribution of the involved nerve.
Loss of normal lumbar curve.
Spasm of the lumbar muscles.
Limited and painful motion.
Listing to one side.
Limping gait.
Limited straight leg raising (LaSegue's sign) causing pain.
Positive Kernig test.
Tenderness in sciatic nerve.
Diminution or absence of Achilles tendon reflex on involved side.
Segmental sensory loss and muscular weakness.

Leaving consideration of these symptoms for the time being, it may be well to examine a cross section of the spine at the lumbosacral level. The lumbosacral joints lie at approximately 45 degrees to the anteroposterior plane of the body; this is the angle which is considered normal. There is, however, such a great variation in the normal that it is impossible to say in any particular case that the articulations are normal or abnormal for that individual. The joints lie in different planes horizontally as well as perpendicularly, and frequently vary in angle in the same person on opposite sides, one being perpendicular and the other nearly or entirely horizontal. Any condition that minimizes the mechanical stability of the lower spine so far as articulation of the bones is concerned, throws strain on the structures which support this region, namely, the ligaments and the joints.

There is no joint in the body which carries as much weight per square inch as the lumbosacral and 4th and 5th lumbar articulations. The entire weight of the upper part of the body in the upright position falls on these facets. When the body is flexed, these facets glide on each other and some of the weight is transferred to the bodies of the 4th and 5th in front, and the ligaments which support the joints and permit motion are put upon the stretch. The integrity of motion depends upon many structures, the interspinous ligament, the joint capsules, the ligamenta flava, and the posterior longitudinal ligament. Flexion of the spine, therefore, not only puts pressure on the intervertebral disks and bodies but also puts stretch on the other tissues just named, and the muscles as well.

We are all familiar with the suddenness with which inflammation may be set up by overstrain of joints which are much more easily examined and more superficial than the complicated structures in the lower back, which are covered deep under the muscles activating movements in the back. In other joints we frequently see acute pain precipitated by rather unforceful motions, twists or wrenches, accompanied not infrequently by swelling not only within the joint cavity, causing distension of the joint, but in the surrounding ligaments as well, causing thickening and induration. Such symptoms are more common in patients with some degeneration of the cartilage of the joint. This degeneration of cartilage and hypertrophy around the edge of the joint can be reproduced in animals, regularly and consistently, by causing an instability and allowing motion and weight-bearing. Cartilage from such a joint, whether it be the knee, hip, elbow or spine, shows the same pathology; a poorly staining, thin, cartilaginous structure, with condensation of nuclei and lack of cylindrical cells at the matrix, the matrix being thin and sometimes completely obliterated at the point of greatest pressure. Degeneration of cartilage implies degeneration of ligaments around the joint. When that occurs the ligaments have lost their elasticity and the joint has lost at least part of its smooth gliding bearing surface. Any joint in which this has occurred will, occasionally and sometimes chronically —depending on the stage of the process—give acute pain and locking; in other joints we can demonstrate accompanying swelling within the joint and in the surrounding structures which further contributes to pain and limitation of motion.

In the lower spine there is no reason to suppose that the joints are not as freely supplied with nerves as other joints in the body, and it is a well known fact that joint pathology can and does frequently give excruciating pain. Jarring, such as caused by coughing or sneezing, which causes sudden contracture of the muscles of the back and abdomen, usually accompanied by flexion of the body, could cause excruciating pain by the sudden jerk of this reflex action. The nerves which exit through the intervertebral foramen bear the same relation to the joints posteriorly as does a ruptured disk anteriorly. The nerve roots exit from the foramen about midway between these two structures in their descent from the dura, so that swelling of the capsule of the joint, or of the ligamenta flava which is continuous with the joint capsule, can impinge from a posterior direction as well as a ruptured nucleus can impinge from the anterior direction. Whether the fifth lumbar nerve is pressed upon as it descends laterally opposite the fourth lumbar disk or as it exits laterally in front of the fifth lumbar joint capsule and ligamenta flava, cannot be diagnosed in my opinion before it is surgically exposed. The posterior longitudinal ligament is rather a strong structure of heavy fibrous tissue, and it would not seem that it would rupture from pressure by the nucleus; rather, the nucleus, a semi-solid gelatinous structure, would be pressed laterally and backward and come around the edge of the longitudinal ligament. This ligament lies anterior to the cord fully three-fourths of the width of the canal at this point; an internal plexus of vertebral veins enters the bone lateral to this ligament, which would add to the weakness at this point whether the disk may wedge the nerve between it and the bony groove through which it descends. The same nerve can be pressed upon by a swelling of the joint a little lower in its course through the canal, which is none too large to accommodate its size.

The fifth lumbar nerve varies tremendously in size and direction at its exit. It sometimes divides and comes out in two foramena divided by a strong band of fibrous tissue, two branches actually passing through the ligament a full half inch apart, the lower branch being rather tightly compressed by a broad ligament. In other cases the foramen is very narrow from top to bottom and rather wide from side to side in comparison; and in still others there is a long narrow opening which varies in the third, fourth and fifth exits of the same specimen. Injection of a joint in a fresh cadaver can be demonstrated to narrow these spaces anteroposteriorly, actually impinging the nerve between the joint and the anterior wall of the foramen.

In all my experience with low back pain accompanied by sciatica, I have seen but two cases in which the sciatica occurred coincident with the pain. In both these cases there was a complete break in continuity at the lumbosacral sacroiliac junction. In one case, pain occurred immediately following fracture at the lumbosacral joint, accompanied by sciatic symptoms, toe drop, motor and sensory disturbance confined to the distribution of the fourth and fifth, and first, second and third sacral nerves. This pain was relieved when the fracture was reduced, although the nerve symptoms did not subside for a number of months.

The other case had complete separation of the symphysis without fracture, but with a two-inch spread between the tranverse rami on the right and left side, with distortion of the lumbosacral and sacroiliac joints. This patient exhibited the same sciatic pain with which I had been familiar in connection with lesser low back injury. It was promptly relieved when, by manipulation, the normal anatomic relationship was reestablished.

The fact that pain in the sciatic nerve does not come on, in most cases, until weeks or months following injury, would indicate that the pressure is a slowly developing process, therefore not due to extrusion of the nucleus or even forceful projection beneath the ligament; but rather a gradual swelling produced by an inflammatory process which is communicated to the nerve. Also, I have watched the development of loss of reflexes over a period of ten days to two weeks in a patient who insisted on continuing work. In this case the arthritis was on an infectious basis, although the first symptoms were in the lumbosacral region. The reflexes did not reappear until months after the pain in the back due to arthritis had disappeared.

Pain in the back which occurs at night and in the early morning is a typical symptom of joint disability, and is found frequently in other joints as a result of stiffening of ligaments after more or less prolonged disuse. It is probably on a circulatory or toxic basis, or both.

What I have said with reference to these two proven factors may be doubted, because I cannot prove either point except by clinical observation and the word of the patient. The problem is now being attacked from the chemical standpoint, although as yet no publication has been made. I have seen these cases clear up entirely with the administration of a sufficient quantity of hydrochloric acid with meals to correct intestinal function. I have also seen them clear up with a change in diet, aimed to lowering protein and purin intake, even though the blood uric acid content was not high.

When one considers all these factors as being possible causes of low back pain accompanied by sciatica, it would seem that not only a most thorough examination of the lower back should be made, but also an examination of the patient as a whole; remembering that the history is as important in this type of case as it is in diagnosis of gastrointestinal and other conditions. One does not casually make a diagnosis of gastric ulcer or carcinoma of the digestive tract on the symptoms of pain alone, and the lower back is as complicated a mechanism as is the gastrointestinal tract.

If there is sufficient degeneration in the structures of the lower back to permit rupture of a disk through its surrounding membranes, it can be suspected that there exists a like amount of degeneration in other structures making up the lower spine; that if a disk is removed, there is removed also part of the supporting structures of the vertebra at that point. This in turn allows the interspace to collapse, and this changes the anatomic relation between the joints of the two vertebrae involved and throws them in a distorted or strained position, which of itself will cause in time strain enough on the joints in this abnormal position to allow further degeneration in the anatomic structures, and recurrence of pain. I have seen enough cases post-operative to disk removal to warrant the opinion that removal of a disk alone, while it may relieve pain, will allow weakening of the structures in the lower back which may in the future cause recurrence of symptoms. It is further my opinion that removal of the disk as such does not account for the relief of pain in these cases, because decompression of the nerves may occur when the ligamentum flava is detached from the lamina and the anterior joint capsule is frequently opened. If it is necessary to incise the annulus or posterior longitudinal ligament by a crucial incision to gouge the nucleus out with a punch, no rupture of the disk exists. If there is a ruptured nucleus it is plainly visible as a protrusion lateral and anterior to the dura, with a nerve definitely pinched between this protrusion and the side of the canal.

Pain in the back accompanied by pain referred down the sciatic nerve is a symptom, and does not constitute a diagnostic syndrome of any one condition which may cause the symptoms. Let us be honest with our patients and ourselves and put an end to the epidemic of operations for ruptured disks, most of which do not exist.

* * *

THE SPEAKER: *Gentlemen, at this time I want to present to you a man you all know who is president of our organization for this year, Dr. Frank H. Lahey, of Boston, Massachusetts.*

DR. FRANK H. LAHEY: Dr. Pfeiffenberger and Members of the International Medical Assembly: I would like you to know that I appreciate the Presidency of this organization; I appreciate it because it has demonstrated that it fills a useful and valuable purpose in this country. This is demonstrated, it seems to me, in two definite ways: (1) Almost every teacher in the United States is willing and anxious to come here and present to you anything he has that he thinks is of value; and (2) you demonstrate by your presence in such numbers when we are all working so hard, your desire to acquire and accumulate further knowledge with which to practice better medicine, both very laudable purposes and principles.

I have been speaking before this organization now for a number of years,—for so many years that I would not care to count them,—certainly almost since its beginning. I have given lectures to you when you really did get up in the morning. You used to start at 7 o'clock; you have become a little modern and now start at 8. I have given lectures before this organization at 7 in the morning and at 9 at night, and always with an interested and adequate audience. Nothing I can say could more eloquently demonstrate the usefulness of this organization or your appreciation of it.

I would like to say that I honestly believe with no flattery that this organization represents in America today, and if in America, then in the world, the greatest post-graduate teaching organization in existence.

DIAGNOSTIC PEDIATRICAL CLINIC

Practical Points in the Diagnosis and Treatment of Infections of the Urinary Passages in Childhood

By HENRY F. HELMHOLZ, M. D.
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It is very helpful in making a diagnosis to know at what age periods the disease occurs with the greatest frequency. Infections of the urinary passages occur in the neonatal, the diaper age and less frequently during childhood. The neonatal infections of the urinary passages may be the cause of fever for which, on ordinary physical examination, no cause is found. In this age period they are as frequent among boys as they are among girls. In the diaper age, after the first few weeks, the disease is found four to five times as frequently among girls as it is among boys. In this period the outstanding finding also is the absence of signs on physical examination, and only fever, pallor and an occasional chill suggest an infection of the urinary passages. Later in childhood, the difference in incidence is again considerably less, although infections in girls still predominate. It is in this last period that the differentiation of the urinary infection with and without obstruction is usually made, or should be made; in many instances, it can be made in infancy.

The diagnosis of pyogenic urinary infection usually is suggested by an absence of findings rather than by positive findings. For example, frequency of micturition in young children, that is the passage of a few cubic centimeters of urine every fifteen to twenty minutes, is practically never a symptom of a urinary infection. Because the infection is a condition in which signs and symptoms are marked by their absence, the positive diagnosis rests solely on the urinary findings.

In making a diagnosis, it is essential to obtain a specimen of urine which is free from contamination by the vagina or prepuce. Too frequently, the admixture of pus cells from a slight vaginal irritation due to fever has given rise to a wrong diagnosis of urinary infection.

Even today, many physicians consider the use of the catheter a dangerous procedure, which should not be carried out routinely. It is interesting to know that in some quarters there is still much opposition to catheterization. Experimentally, it is practically impossible to infect the normal animal bladder by the introduction of gram-negative bacteria. If one goes back into the literature, one finds that, in 1883, as excellent an observer as Hirschsprung, of megacolon fame, stated that all patients admitted to his service from whom a specimen of urine could not be obtained immediately, were catheterized and in no case did he see untoward results in a series of many thousand cases.

There is just one condition in which the introduction of a catheter into the bladder is associated with danger, namely obstruction at the vesical neck or in the urethra. With residual urine in the bladder, the introduction of even a few bacteria may set up a serious infection.

DIAGNOSIS

The obtaining of a specimen of the bladder urine free from contamination by the vagina or prepuce is essential. To obtain such a specimen is relatively simple if the patient is a boy. The prepuce is retracted, the glans penis is washed off with a little boiled water or tap water, and the boy is asked to micturate into a pan. After he has passed a few cubic centimeters of urine, a specimen is caught in a sterile test tube. This is then examined for pus and for bacteria. In examining for bacteria a smear of the urinary sediment is stained by Gram method or a culture is made in a tube or on a plate. If the patient is a girl, it is possible to obtain an uncontaminated specimen of urine by spreading her legs apart, cleansing the urethral opening carefully with cotton that has been moistened with sterile water or a 0.5 per cent solution of compound solution of cresol, and asking the patient to micturate into a pan. It is possible in this way to catch an uncontaminated specimen of urine. It is simpler and less time-consuming to catheterize the patient. For this purpose, short glass catheters, 10 cm. long, to which about 4 cm. of rubber tubing has been attached, are put up sterilely in test tubes that can be used to hold the specimen of urine.

In the office or hospital, the specimens that have been obtained under sterile precautions can be cultured on eosin methyl blue and blood agar plates to identify the infecting bacterium. The sediment of a centrifuged specimen of the urine can be spread on a slide, fixed and stained by Gram's method. This will distinguish gram-negative from gram-positive bacteria, which is very important.

In the home, only a catheter and a tube of agar are necessary. The catheter is boiled in a pan and, on cooling, some of the boiled water is used to cleanse the external meatus. The agar tube is melted and cooled so that it is comfortable on the cheek. It is inoculated with 3 drops of urine and incubated in the vest pocket for twenty-four hours. The sediment of a catheterized specimen can be examined for bacteria and pus directly under the microscope or after it has been stained.

Escherichia coli, Aerobacter aerogenes, Proteus vulgaris and Pseudomonas have a characteristic appearance when grown on eosin methylene blue agar. More important from the therapeutic standpoint is the Streptococcus faecalis, which is the only gram-positive streptococcus that will grow on this medium. One can be quite certain, therefore, that a gram-positive coccus growing on an eosin methylene blue agar plate is the Streptococcus faecalis. One may ask whether it is necessary to determine the kind of infecting bacterium. Before the advent of the sulfonamide drugs it made little difference what the cause of the urinary infection was, because all urinary infections were treated alike. The resistance of the various bacteria to therapy did not vary a great deal, except in so far as it was difficult to acidify the urine when the infecting bacterium alkalinized the urine.

The ease of administration and effectiveness of the various sulfonamide compounds in cases of urinary infection with the gram-negative bacteria has led to the routine use of these compounds in all urinary infections. I showed, in my first publication on the use of sulfanilamides, and later in articles on the other sulfonamide compounds, that the Streptococcus faecalis is not killed by the drugs and, in fact, grows luxuriantly in concentrations of the various sulfonamides that sterilize a urine infected with Escherichia coli within twenty-four hours. It has been my experience repeatedly that in cases of infection in which a primary culture discloses only Escherichia coli, a pure culture of Streptococcus faecalis may be obtained after two days of treatment with one of the sulfonamides. The following case shows the necessity of identifying this infecting bacterium by culture.

A patient who was operated on for a large ureterocele returned home against the advice of her surgeon. Her urine still contained blood, pus, and bacteria. For one year the patient was treated with various sulfonamides without any effect, and because of this was considered to be in need of further operative procedures. Culture of the urine disclosed Streptococcus faecalis. Cystoscopic examination revealed that the bladder and ureters were normal. The infection was cleared up in the course of the next ten days by means of mandelic acid therapy. A culture taken many months previously would, in all likelihood, have brought about a change in drugs and a rapid cure of the infection.

Having established the presence and the nature of the infection, one must determine the state of renal function and presence or absence of obstruction in the urinary passages.

In the average case in which a child has a urinary infection that clears up rapidly under drug therapy, it seems hardly necessary to determine the concentration of blood urea, to do a phenosulfonphthalein test, or to make an excretory urogram unless the infection recurs or persists. The more experience I have with abnormalities of the urinary passages, which occur in about 7 per cent of all persons, the more I am inclined to make an excretory urogram as a measure of safety for the early recognition of either obstruction or stasis in the urinary passages. There are still too many infants, children and adults dying of renal failure who might have been saved by early recognition of the obstruction and infection of the urinary tract. The diagnosis of urinary infection is largely a matter of obtaining an uncontaminated specimen of urine and examining it with the microscope and by culture.

TREATMENT

It might be of interest to digress just a minute to say a few words regarding the history of various drugs that have been used to combat urinary infection. Bokai, in 1878, recommended alkalinization with sodium bicarbonate in cases of infection in which the urine was acid, and acidification in cases of ammoniacal cystitis. Even now some physicians consider alkalinization of the urine the treatment of choice of urinary infection in children. About all that can be said for the use of alkalis is that they may act as diuretics. It is of practically no value compared with the other drugs which have been introduced more recently. Methenamine, which was introduced by Nikolaier in 1894, probably was the best of the urinary antiseptics in use before the ketogenic diet was introduced in 1931. The introduction of beta-oxybutyric acid was followed rapidly by the introduction of another organic acid, mandelic acid, by Rosenheim in 1935, which made it possible to administer an organic acid by mouth instead of using the ketogenic diet to produce it in the body.

It will be noted that methenamine, the ketogenic diet and mandelic acid all require a definite degree of acidity of the urine for the development of their action. The necessary degree of acidity seems to be about pH 5.5. The attainment of this pH is the stumbling block in many cases of urinary infection. Two distinct groups of cases are recognized: (1) those in which the kidney is damaged and cannot excrete the acid in sufficient concentration; and (2) those in which the infecting bacterium produces an alkalinity that persists in spite of the administration of acids and acid-forming salts.

TABLE 1.

SULFATHIAZOLE AND SULFADIAZINE

Condition necessary for bactericidal action of urine:

Concentration of 100 mg. per 100 c.c.

Dosage:

½ grain (0.032 gm.) per pound (0.45 kg.) of body weight in 24 hours.

Given in 4 doses: after meals and at bed time. Adult dose 30-45 grains (2-3 gm.).

Procedure:

1. Keep urine sterile for 4 to 6 days.
2. Culture urine for sterility not less than 4 days after discontinuing use of drug.

The introduction of the sulfonamides in 1936 was a great advance, because these drugs act in both acid and alkaline urine and are excreted in the urine by the damaged kidney in bactericidal concentrations. The sulfonamides thus fitted right into the weakest spot in our armamentarium in the treatment of urinary infection. The only weak spot of the sulfonamides is their total lack of effect on the Streptococcus faecalis, and on this hinges the important reason for cultures of the urine in the treatment of urinary infection.

TABLE 2.

INDICATIONS FOR USE OF SULFONAMIDES AND MANDELIC ACID OR METHENAMINE

Successful therapeutic results with sulfonamides can be expected in all simple infections except those caused by the Streptococcus faecalis.

For infections of the urinary passages caused by Streptococcus faecalis, mandelic acid or methenamine must be used.

In order to simplify the choice of a given drug, and its dosage to obtain the necessary concentration in the urine, I have made a series of tables which are practically self-explanatory. They give the simplest routine procedure and pass on to the exceptions and variations of therapy when the simpler ones are not successful.

The treatment which is the simplest, should be the treatment of choice. The sulfonamides are selected because they are very unlikely to produce any nausea or renal damage when administered in doses necessary for urinary antisepsis. The dose of these drugs is small, the reaction of the urine does not need to be taken into account, and the drugs are excreted by the damaged kidney in bactericidal concentration.

Table 1 shows the mode of procedure with either sulfathiazole or sulfadiazine, which so far have appeared to be more effective in lower concentration than have the other members of the group. Emphasis should be placed on the determination of urinary sterility after discontinuing administration of the drug.

TABLE 3.

URINARY INFECTION WITH STREPTOCOCCUS FAECALIS CURED WITH MANDELIC ACID AFTER FAILURE WITH SULFANILAMIDE

Day	Treatment	Dose		Pus, grade	Sulfanilamide, mg. per 100 c.c. in urine		Organisms per 0.5 c.c. of urine
		Grams	Times per day		Free	Conjugated	
1	Sulfanilamide	0.33	6	3	—	—	Innumerable
2	Sulfanilamide	0.33	6	1	—	—	900
3	Sulfanilamide	0.33	6	1	52	31	Innumerable
5	Sulfanilamide	0.33	6	1	98	68	Innumerable
6	Ammonium mandelate	1.0	4	1			Innumerable
9	Ammonium mandelate	1.0	4	0			0
11	Medication discontinued			0			0
14	None			0			0
20	None			0			0

Table 2 emphasizes the one great drawback of sulfonamide therapy, that is its ineffectiveness against the Streptococcus faecalis. Table 3 illustrates the failure of sulfanilamide therapy and the rapidly

TABLE 4.
ADMINISTRATION OF METHENAMINE

1. Render urine acid with ammonium chloride 0.5 to 1.0 gm. four times a day.
2. After 24 hours, test pH of urine (nitrazene paper).
3. If pH is 5.5 or below, give 0.3 to 0.5 gm. methenamine every 6 hours.
4. After 48 hours culture urine.
5. If not sterile, increase each dose by 0.15 gm. every fourth day up to tolerance, as shown by urinary frequency and hematuria.
6. Culture urine for sterility four days after discontinuing administration of drug.

successful treatment of a patient with a Streptococcus faecalis infection. Table 4 shows the mode of procedure for treatment with methenamine. Table 5 shows the mode of procedure for treatment with mandelic acid.

Patients with markedly impaired renal function cannot excrete mandelic acid or methenamine in bactericidal concentrations but can excrete the sulfonamides in such concentrations. When the renal damage is unilateral, the vesical urine may become sterile as the result of excretion of a strongly bactericidal urine from the normal side, but an immediate relapse may occur when treatment is discontinued.

Table 6 shows the very remarkable effect of the administration of about 1 grain (0.065 gm.) of sulfathiazole a day in rendering a markedly cloudy urine crystal clear, and in decreasing the number of bacteria. To be emphasized is the fact that the infection was due to Pseudomonas, which usually is very resistant to therapy. The concentration of blood urea was more than 200 mg. per 100 c.c.

Finally, it is important to emphasize the need of further work with new compounds to fill in the gap in the successful treatment of damaged kidneys and a Streptococcus faecalis infection.

The preceding six tables illustrate the treatment in the average cases of urinary infection in childhood; if the treatment is not rapidly successful, and in all cases with lowered renal function or obstructions in the renal passages, the help of the urologist should be sought immediately, so that obstruction in the urinary passages can be eliminated at the earliest possible moment.

The early recognition of urinary obstruction may be life-saving.

TABLE 5.
USE OF MANDELIC ACID

Conditions necessary for bactericidal action:
1. 0.5 to 1 per cent mandelic acid in urine.
2. pH of 5.5 or less (nitrazene paper).

Dose:
1 gm. ammonium or calcium mandelate in 24 hours for each 100 c.c. of urinary output.
Adult dose: 3 gm. 4 times a day after meals and at bed time.

Procedure:
1. Keep urine sterile for 4-6 days.
2. Culture urine for sterility 4 days after discontinuing administration of drug.

TABLE 6.
STRIKING EFFECT OF SMALL DOSES OF SULFATHIAZOLE IN CONTROLLING PYURIA ASSOCIATED WITH HIGH CONCENTRATION OF BLOOD UREA

Boy, 16 years of age. Left Clinic 2-24-41. Taking 0.06 gm. twice daily.
Urine clear until stopped drug 3-21 because of a convulsion.

		Urine			Dose		
Date, 1941	Blood urea, mg. per 100 c.c.	Pyuria, grade	Bacteria, per 0.5 c.c.	Sulfathiazole, mg. per 100 c.c.	Gm.	Total daily, grains	Remarks
3-24	222	4	Innumerable		0.012 x 5	1	Pseudomonas infection
3-27		4	200	1.0	0.012 x 3	3/5	No medication from 10 P.M., 3-26 to 4 P.M., 3-27
3-28	222	2	400	1.0	0.012 x 5	1	Urine cloudy
3-29		1	550	1.0	0.012 x 5	1	Urine crystal clear

DIAGNOSTIC PSYCHIATRICAL CLINIC

Recognition and Management of Acute Psychoses in General Practice

By FRANCIS J. GERTY, M. D.
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I believe it is fair to assume that the practice of psychiatry is generally regarded as including patients who can be classified from the standpoint of practical diagnosis of their cases into two groups: those who are psychoneurotic and are treated in the office, and those who have psychoses and should be managed intramurally or within the confines of an institution for mental patients.

The general practitioner has a large share of the psychoneurotic patients, many of whom will be referred to the psychiatrist when they become too bothersome or when the resources of physical treatment have been pretty well exhausted. As to the psychotic group, the general practitioner usually considers the patient as being outside of his province from the very beginning. If in the course of some illness, either medical or surgical, the patient shows evidence of being psychotic, there is commonly a choice between two methods of managing the case: either sedatives are used for quieting the patient to the point of unconsciousness, or the patient is promptly routed to a psychiatric institution, usually a sanitarium or psychopathic hospital. Ordinarily general hospitals make no provision for caring for such patients in a separate psychiatric unit. There are exceptions to this, of course, but the exceptions are relatively few. It is unfortunate that so often the general practitioner or the internist, surgeon, obstetrician or gynecologist loses all touch with the treatment of patients who become psychotic. It is also unfortunate from the standpoint of the patients that they commonly lose the benefits which certainly would be theirs were their care continued in the general hospital with all of its modern facilities for diagnosis and treatment.

Some of our psychiatric hospitals have such facilities available, but this is not often the case. The principal bad effect of our present method of shunting psychiatric patients out of view into the confines of a purely psychiatric hospital has resulted in insufficient knowledge on the part of practitioners of medicine of the possible connections between human behavior and physical illness. From the patient's standpoint the benefits to be gained by other than purely psychiatric procedures are often sacrificed.

Actually, the general practitioner finds many psychoneurotic patients included in his practice and he does see a certain number of psychotic patients.

The psychotic patients fall into two groups: those who are seen by the physician once or twice before they are committed for a probable long-term treatment in a psychiatric institution because their problems are chiefly psychiatric; and those who will probably require only short-term treatment, either because they have acute psychoses of the more common sort, such as manic-depressive or schizophrenic psychosis, or because their psychosis complicates some physical illness. Improvements in methods of handling patients in the latter group often make it possible to avoid commitment to a psychiatric institution entirely. It may be of interest to you to know that on January 1st the new Mental Health Act goes into effect in the State of Illinois. It will permit the treatment of patients with short-term psychiatric illnesses without commitment. Psychiatrists feel that this is a very great advance.

It is suggested that for the purposes of such short-term treatment the psychiatric unit in a general hospital is probably the best method of management. The establishment of such units will also give the general practitioner the opportunity for collaboration in the intelligent treatment of patients whose psychoses arise in connection with medical and surgical conditions.

Patients with the ordinary, old-time, recognized forms of psychoses, and those in which the psychosis is connected with physical illness, should not be handled as purely psychiatric problems in a purely psychiatric institution.

It is hardly necessary for me to remind you of the applications of fever treatment in general paralysis of the insane. I do not intend to go into the relative merits of various types of fever treatment; neither shall I burden you with discussion of the symptoms and diagnosis of general paralysis of the insane. These subjects are adequately taught in our medical schools, but I would draw your attention to the extreme importance of making an early diagnosis in the case of general paralysis. The fever treatment cannot restore destroyed brain cells, but it can arrest destruction, and the treatment results should yield a very high percentage of good results if the condition is recognized early.

During the past ten years you have also heard a great deal of shock methods of treatment for the so-called constitutional functional psychoses, such as the manic-depressive and schizophrenic types. Usually we think of the treatment of psychosis only with reference to cases of this sort, and the establishment of psychiatric units in general hospitals has been urged on hospital boards only for the purpose of treating such patients. Naturally, such boards have been slow to establish units of this sort which are obviously psychiatric and which will require installation of special facilities. Usually it is thought that such treatments could be managed as well in a psychiatric institution.

Psychiatrists in consulting office practice find that many patients whom they are called to examine do not belong in the category of purely psychiatric problems. An imposing list of cases could undoubtedly be assembled from the files of any practicing psychiatrist in which the physical relationships to the psychiatric disturbances (either in a causative or in a complicating manner) can be demonstrated. I do not intend to review this list in its entirety, but I would point out to you that psychosis can occur in patients under medical management. This is the case in surgical conditions such as head injuries and infected wounds, tumors, and in operative removal of pelvic organs. It is also true in cases of medical conditions such as acute infective diseases, debilitating illnesses, and very commonly in toxic and exhaustive conditions.

Gynecologists and obstetricians encounter mental disturbance in connection with menopausal difficulties and in the puerperium. When such disturbances first come to attention, sedative medication is commonly used, and unfortunately this sort of treatment may possibly increase the degree of mental disturbance. We would do well with more cooperation and understanding between psychiatrists and physicians in other lines of practice and with an extension of the facilities available for the proper management of such patients.

Today I am presenting the cases of two patients through which I believe some of the points about which I have just been speaking will be made somewhat clearer.

The first patient is a forty-three-year-old woman, Jewish, who was admitted to the Illinois Neuropsychiatric Institute on August 22nd of this year. She was well until the last week in July, one month after a hysterectomy. She had had three previous states of melancholia in her life, one associated with the birth of each of her children. When seen on the ward the patient was withdrawn, quiet and mute. She would sit quietly in the corner of the ward, staring into space. When she would respond to questioning she was confused and disoriented. She thought she was being influenced by everyone around her and that people were making fun of her and attempting to harm her. Her appearance was untidy and disheveled and she seemed to take no interest in keeping herself neat.

As to the onset and course, the patient was fairly normal until after the hysterectomy the last week in June. She then became overly conscientious and active. She wanted to get a job. She had had an arrangement with her husband whereby she would meet him at work every Friday noon to get his weekly check from him because he had an unfortunate habit of gambling if she didn't do this. The last week in July the patient was unable to go down on Friday to get her husband's check because the son was sick with tonsillitis. He had a high fever and she thought she should not leave him. Her husband didn't come home either that night or the next two nights. She began to worry not only about her husband but also about where she would get the money with which to pay the rent. The landlord had been demanding the rent for about a week and the patient had told him that she would give it to him on Friday. When he came, however, she had no money with which to pay him. She was unable to sleep that night and complained of restlessness and loss of appetite. She became so disturbed that she could no longer do her housework. She began to express suspicions that people around her were going to harm her. Her husband took her to Dr. George B. Castle, who recommended hospitalization.

The patient's birth and development were normal. She was born in Russia and at the age of twelve had come to Canada with her family. After finishing the eighth grade she received some training as a stenographer and worked for several years. She removed to St. Louis from Winnipeg when she was twenty. Her husband followed her to St. Louis; he was courting her at the time, and at the age of twenty-three they were married. Within a year she became pregnant. She had not wanted the baby and did everything she could to have an abortion. The child that was born was weak, pale and scrawny. She became very much depressed because she felt she had harmed the baby in some way. She became withdrawn at this time and expressed ideas of guilt and sinfulness. After the birth of a second child five years later, whom the patient also did not want, the patient once again had a depression. When she was thirty-six, another child was born, and though this child looked very healthy and she wanted this child, she was afraid when she looked at him that he would be crippled or maimed and she became depressed once again. Then came the present attack after a hysterectomy.

After six electric shock treatments the patient's condition has improved very much. (Patient presented)

Naturally, this patient is somewhat concerned about being before so large an audience. She kindly consented to come down here and I don't want to embarrass her any more than is absolutely necessary.

These are all doctors who have come to see you and they want to know how you have been getting along. Do you feel any better at all than you were?

PATIENT: I don't feel so well yet.

DR. GERTY: She doesn't feel so well yet, she says. From each of these attacks she has made a recovery in a relatively short time and has gone on and attended to the work of her home.

Were you ever sick in this way except when you had a baby or after you had that operation?

PATIENT: I was sick before that.

DR. GERTY: I mean with this kind of trouble, with being melancholy and depressed?

PATIENT: I was before.

DR. GERTY: From the standpoint of presenting her troubles to you in such a way that she can discuss them and make them clear herself, I think probably these are not the right circumstances under which to bring out our point. That is why I have read her history first. We expect to get a recovery in this case. This patient is not done with treatment. She is still in the hospital; she is at least in condition to come down here, although naturally she is embarrassed; she has never been before such an audience before, I am sure.

As I said, unquestionably there will be a recovery from this attack. I cannot of course prophesy as to the future with reference to the possibility of further difficulties of a psychiatric nature. It is also apparent from her history that there are some factors which we must regard as psychogenic, that is having to do more with her experience and relationships with people rather than physical illness.

The second patient is a man thirty-nine years of age who had had four episodes of bleeding gastric ulcer. He has also had some psychiatric disturbance aside from the times that he has had the bleeding gastric ulcer. He entered the Illinois Neuropsychiatric Institute on August 8th from the Psychopathic Hospital in Chicago. His physical condition was poor. He was thin, six feet four inches tall and weighed at that time about 147 pounds, some 40 or 50 pounds less than his normal weight. He was anemic; his hemoglobin at the time was 70. A trace of blood was present in his stools for the first few days. Mentally he was completely disoriented, confused and delirious. Unless restrained and heavily sedated he would attempt to get out of bed and attack the ward personnel. He was quite combative and when approached by a nurse or doctor would spit at them and call them names. He talked rapidly in a disorganized way. For example: "Who are you? Who am I? Are you a relative of mine? Am I German? Am I Hitler? Am I out of order?"

This patient has been working for a public utility company for twenty years. He has been given leave of absence on four occasions for illness. On three of these occasions before the present attack he has been psychotic. Two weeks before his admission to the Institute the patient began to vomit blood. He was taken to the Ravenswood Hospital and was given a blood transfusion. The day after the transfusion the patient became delirious, disoriented and combative. He could no longer be managed, so his transfer to the Psychopathic Hospital was made.

He is the fifth child in a family of seven. His father, a cooper, had difficulty in supporting the family. The patient's childhood was spent in a home overshadowed by impoverishment and financial worry. The father drank a good deal and was abusive to the family when he was intoxicated. The patient left high school when he was seventeen years old, came to Chicago from a nearby town and began to work to help support the family. At this time his parents separated. When he was nineteen he started to work with a large company for which he still works. He has continued in this employment to the present time and is still an employee of the company, though on leave now.

He has had four mental breakdowns, from all of which he has recovered in three months or less. He says that his symptoms have been quite similar in all of the attacks, though the apparent precipitating cause has not always been the same.

Briefly, the story of these breakdowns is as follows. In 1928 when he was twenty-four years of age he began to be tense, agitated and depressed. He had been getting along fairly well in the employment which he had then followed for five years, so well that he worked unusually hard to make progress. Effort and hours meant nothing to him. Then the symptoms appeared. He was cared for in a private sanitarium in the city for two weeks but complained so much of this place that he was removed to another sanitarium in Milwaukee and remained there three weeks. He was intensely bothered about the stigma of being kept in a sanitarium, and prevailed on his family to take him home, though he was not yet well. (Patients of this kind naturally resent being classified in an institution for purely mental patients. I think that is understandable.) He was home for a few weeks and recovered. Eleven weeks after the onset of his illness he was back at work.

In 1939, eleven years later, at the age of thirty-five, he had a second attack which seems to have been precipitated by physical illness. He had a recurrence of bleeding ulcers at this time. He became very weak and anemic and had to be hospitalized for a blood transfusion. Just after receiving the transfusion he became psychotic. He was cared for in a sanitarium for a month, recovered, and was able to return to work. He states that he had a period of reaction after the depressive symptoms had disappeared, in which he was elated and bought things too freely, but this reaction soon subsided. We often notice such things with depressed patients when we have them to the point where they seem to have made a recovery.

His third attack occurred in 1942 when he was thirty-eight years of age, without preceding ulcer symptoms. He began to feel depressed and to have headache. He went to the Mayo Clinic for a period of observation, but nothing significant was discovered. A week after his return he began to read the Bible, talk to himself as being Christ in the second coming, and was bothered by the thought that window screens were charged with electricity. He was in a highly disturbed state of physical activity, was taken to a psychopathic hospital and after one week was sent to a state hospital from which he was discharged within six weeks. He remained home only three weeks longer and then returned to work.

The fourth attack was associated with hemorrhage from ulcers and blood transfusion and began in August, 1943. He is just recovering from this attack and the presenting symptomatology and course of this attack have already been discussed.

I wonder if you would tell the doctors how you feel.

PATIENT: You want me to tell them how I feel?

DR. GERTY: Yes, and what you think of the attack that you had this summer.

PATIENT: It seems I get these psychiatric symptoms mainly after blood transfusions and I feel all right now mentally but I am very weak physically.

DR. GERTY: He does have some neuritic symptoms remaining. Do you think you will be able to get back to work shortly?

PATIENT: I won't be able to do the same type of work I have been doing until I recover more than I have.

DR. GERTY: As to the symptoms which you have had, can we frankly term them mental symptoms? Do you agree to that?

PATIENT: That is right.

DR. GERTY: You understand that most things were not normal, that you did not have grounds for the beliefs you expressed?

PATIENT: No, evidently they were delusions.

DR. GERTY: If time permitted and if it were within the province of this presentation we might discuss the possible psychogenic factors in his attacks. They require only brief mention for our present purposes. The privations and unpleasant experiences in his early family life may have some bearing. He is Jewish and though happily employed by a company which does not have a great many Jews working for it, he nevertheless suffers from hurt to his racial pride when he overhears discussions, particularly because many people do not think that he is Jewish and sometimes they do not refer to Jews in a complimentary manner. He is married to a Gentile girl, has been married six years, and his family, especially his mother, were not really happy about this marriage, which took place only after four years of acquaintance. He gets along well with his wife but there is some strife because of the family's unwillingness to accept the union fully. However, though his background might suggest predisposition to psychosis of a psychogenic sort, we cannot dismiss very lightly the immediate effect of the blood transfusions which were followed by such acute mental symptoms.

As to his management, after the stools were blood free he was put on a high caloric, high vitamin, bland diet, and has since gained ten pounds. The blood picture is now improved. The mental condition did not change with the physical improvement at first. After one electric shock treatment, however, he became rational, oriented and cooperative.

Much might be said with regard to the cases of these two patients and concerning the whole group of acute psychoses, especially those acute psychoses in which physical illness seems to play some part.

The field can be explored with profit, I am sure. We may speculate as to the role of all sorts of predispositions to psychosis, starting with heredity and constitution and following the matter through all of the intricacies of psychically traumatic life experiences from early childhood. From predisposition we may go on to exciting and precipitating factors of apparent importance in the single attacks of acute psychotic disorder. In some instances these factors will seem to be chiefly physical; in some chiefly mental. The practitioner who takes a wide and objective view of his patients' troubles will probably find admixtures of these factors in greatly varying proportions. Why, then, should either set of factors be at all neglected? Why should the physician who is called a psychiatrist tread a way so different from the physician who is called a surgeon, an internist or obstetrician? Much of our practice, and especially our hospital practice, seems organized on this sort of separatist principle; yet none of us truly believes our patients are so nicely partitioned as to body and mind, however much we must make use (for simple clearness and convenience) of the separate approaches in our slow gathering of information. We should remember that the facts gathered must be reassembled, synthesized and integrated if we are to understand our patient as a living individual.

In closing, I would state these truths which appear from experience:

1. Many psychoses are recoverable, often in a relatively short period of weeks and months. It is unfortunate that patients who make recoveries in such short time should sometimes be adjudged insane.

2. Many psychoses have highly important physical aspects, both in cause and effect.

3. We have been inclined, with great injustice to the patient and with detriment to medical progress, to follow separate paths, psychiatric in the sense of being mental, medical in the sense of being physical. This has minimized discovery and understanding, and, as a consequence, progress in treatment of mental disorders has been hampered.

4. Our thinking and our practice and also our facilities should be changed or organized so that we meet the problem of mental disorder as it actually exists.

THE SPEAKER: *We will now have a diagnostic clinic, Parkinson's Syndrome and Chronic Encephalitis, by Dr. Oscar Hawkinson, Neurologist, St. Anne's and West Suburban Hospitals, Chicago, Illinois; President, Chicago Medical Society. Let's give him a rising greeting because as Chairman of our Chicago Committee he has done a wonderful job.* (Applause) *We have had splendid cooperation from the Chicago doctors, and the Chicago Medical Society in particular. I take great pleasure in presenting Dr. Hawkinson.*

DIAGNOSTIC NEUROLOGICAL CLINIC

Parkinson's Syndrome and Chronic Encephalitis

By OSCAR HAWKINSON, M. D.
Neurologist, St. Anne's and West Suburban Hospitals, Chicago, Illinois;
President, Chicago Medical Society

Mr. Chairman, Ladies and Gentlemen:

It has been a great pleasure for the men of Chicago to work with the Interstate group. It has been a real pleasure, and any time that we can do anything to help a project of this kind we are happy to do so.

It was my thought that there might be a few in this Assembly who would be interested in a brief review of Parkinson's disease.

Parkinson published his essay on "The Shaking Palsy" in 1817 when methods of investigation and research were rather primitive, when the trained pathologist with microtome and microscope was almost unknown. The author, by way of apology, states that analogy and conjecture take the place of exact knowledge gained from anatomical studies and he hopes that his report may stimulate further inquiry.

Parkinson reasoned that since the senses and the intellect were not impaired the pathology did not extend to the encephalon and it was his opinion that he had demonstrated absence of peripheral nerve lesions and therefore the disease must be found in the medulla spinalis. He reasoned further that this part of the cord being in the freely movable portion of the spinal vertebrae would be more prone to injury.

While Parkinson published his essay on "The Shaking Palsy" in 1817, the term "paralysis agitans" was first used by Marshall Hall in 1841. Causes of this malady as described by Parkinson were those that the older men in this room learned when medical students: worry, grief, shock, exposure to cold, overwork, excesses of various kinds, arteriosclerosis, syphilis, and so forth. He spoke of a patient who had suffered with rheumatism which might possibly have been a causative factor. These we all were taught to believe were active in the causation of paralysis agitans.

Since the epidemic of encephalitis in 1917 and 1918 came many opportunities to study the chronic cases with their marked resemblance to paralysis agitans, much has been learned about its pathology. Indeed, the pathology of the two conditions as well as the clinical manifestations are so similar that it is not strange that question as to the occurrence of two distinct disease entities has been made.

Parkinson's definition continues to be a classic. He says, you remember, that paralysis agitans is a nervous affection characterized by involuntary tremulous motion with lessened muscular power in parts not in action, and even when supported, with a propensity to bend the trunk forward and to pass from a walking to a running pace; the senses and the intellect are not impaired.

As to the etiology, omitting those cases which occur as a result of encephalitis, paralysis agitans must be considered a rare disease. Eulenberg reports in a series of 10,424 cases of nervous affection, 46, or 0.4 per cent. Krafft Ebings' series of 34,000 showed 68, or 0.2 per cent; Mendel in 25,000 cases reports 50, or 0.2 per cent; while Erb reports 78, or 0.7 per cent in a series of 11,049 cases.

The occurrence in men is slightly more frequent than among women in a ratio of about 10 to 7. In older age groups, after 65, the proportion is about equal. Before 1910 only fourteen cases were reported by Willege whose onset occurred before the age of thirty. Since that time, so-called juvenile cases are reported with onset as early as six or seven.

Von Bogaert had a case occurring at the age of seven which died at the age of thirty.

Predisposing causes are those mentioned by Parkinson and have already been noted: fevers and infections are ascribed as causes if in no other way than the lowering of resistance; gout, typhoid, influenza, malaria, barbital poisoning, manganese, carbon disulfide; trauma has been discussed as an etiological factor. While many authorities are inclined to place little or no significance on trauma as a cause, it is my belief that it has influenced a great many. Family tendencies have also been noted by some authorities. Weber had a father and son; Gowers had a mother and son; Stewart, three cases, a maternal aunt, paternal grandfather and a sister.

While, as stated, paralysis agitans occurs infrequently, Parkinsonism following epidemic encephalitis is quite common, occurring sometimes a few weeks, at other times several years, after the acute attack. Since the influenza epidemic of 1917 and 1918 and again in 1925, many of these cases are seen, occurring in all age groups. In some epidemics as many as 30 to 35 per cent of those who survived have suffered involvement of the basal ganglia and in the course of time developed Parkinsonism. One observer reports a case developing eleven years after the acute attack. In not a few instances Parkinsonism may occur without the observer being able to secure definite information relative to any illness that might have even remotely any relation to his malady.

There seems to be a wider variation in the symptomatology in postencephalitic Parkinsonism than in Parkinson's shaking palsy. In children, we frequently find marked personality changes; muscular rigidities may occur on one side while the contralateral side is flaccid; choreiform movements occur sometimes so large and expansive as to border on the grotesque. These release phenomena are sometimes so difficult to control that the sufferer must pause until the spasm ceases.

Sialorrhea also seems more troublesome. In those more nearly resembling paralysis agitans there is found muscular rigidity, immobile face, some mental depression, festination, diaphoresis, and the characteristic tremor.

Violet headaches sometimes occur, and sometimes we have a great deal of insomnia.

As to pathology, the pathology is found principally in the subthalamic region and consists of a chronic degeneration of cells in the corpus striatum along with the efferent fibers. The globus pallidus is much involved. There is secondary degeneration of the body of Luys. The ventricular body undergoes degeneration and atrophy. The normal structures disappear and are replaced by glia cells and fibers. The blood vessels show no pathologic changes that are unlike those of the surrounding structures. The pathology which follows Parkinsonism after epidemic encephalitis differs very little from that just described; practically the same structures are involved and produce the same release phenomena that we find in paralysis agitans. There is atrophy of neuron and proliferation of glia cells. After about three and one-half years, according to Renaud, there is no trace of inflammatory process and only evidence of degenerative changes remain.

Of the nature and pathogenesis, Wilson and Bruce in their recent work have this to say: "What appears most likely is that the inherent weakness of certain striatal cell fiber systems brings their span of life to a premature end when involution begins and tends to continue with hardly a break. Should the disease last long enough, senility and vascular degeneration will add their combined quota to the clinico-pathological sum."

As to diagnosis, in the early stages diagnosis is not so simple, but after a year or maybe sixteen or eighteen months it is rather easy. The immobile face, postural changes, increased muscle tone and characteristic tremor sometimes enable you to make a diagnosis when the patient walks into your office.

There are no pyramidal tract signs and very few sensory disturbances.

Senile tremors are usually limited to the head and sometimes you may be fooled by calling it just a senile tremor and in maybe a year or two you have tremors of the hand and the immobile face and other symptoms that come with paralysis agitans.

In multiple sclerosis there are the characteristic remissions and definite involvement of the pyramidal tract. If at any time there are changes in the motor reflexes in paralysis agitans they would always find their way to the motor tracts.

Tremors of psychoneurosis should not offer much difficulty.

General paresis may be ruled out by mental signs and a study of the serology.

Spastic paraplegia has a shuffling gait and motor tract signs.

Chorea in children may sometimes prove troublesome, and sometimes the diagnosis will be made by therapeutic test. If your patient gets well or gets better, which the choreic almost invariably does, you have chorea; if he doesn't get well, you probably have a chronic encephalitis.

I have four patients that I would like to present to you this morning. Two of them are cases of paralysis agitans and two are chronic encephalitis with Parkinsonism.

This gentleman is age fifty-nine. He is a manufacturer. His family history has in it nothing significant. Medical history shows only the usual childhood diseases; no operations; no history of illness since childhood except a neuritis of the left arm in 1927 which soon responded to treatment. No history of lues.

He complained of muscular difficulty, some tremor, inability to perform finer movements of the fingers; some unsteadiness. This was very early. The neurological tests show all motor reflexes present and normal; no abnormal reflexes; eye-grounds normal; sensory field negative; tremors more noticeable in the left than right; heart and lungs normal; blood picture normal; blood Wassermann negative, no study of the spinal fluid was done and no blood chemistry was done; the kidney function was normal; blood pressure 140/85; no arteriosclerosis evident, and little change in the patient's strength.

You notice a quickened knee-jerk and that is all. We used to say that these cases of paralysis agitans never smiled, but I think that is not altogether true. I have two or three doctor friends who have paralysis agitans and they smile a good deal and have made up their minds to take care of themselves, making the best of a hard situation.

This gentleman is a clerical worker, age sixty-two. He was first examined in March 1942. The family history shows nothing significant. He is married and has two children. In the medical history he has had children's diseases; hernia repaired at age thirty; present trouble began about four years ago. At that time he complained of muscular rigidity, vertigo, headaches, regional paresthesia, particularly in the back, and a very troublesome diaphoresis. He walked with marked stooping forward of the body, arms held in rigid flexion; fixed facies; all muscles of the body held in rigid attitude.

The neurological examination revealed all motor reflexes present and exaggerated; no abnormal reflexes except a slight right ankle clonus, the sensory field is normal, vibratory sense normal, and eye-grounds normal.

Again the spinal fluid was not studied and no blood chemistry was done. The blood pressure has ranged over the last year and a half from 120/75 to 140/85, so there is no indication of arteriosclerosis. Blood Wassermann negative; urine negative; no change in one and a half years. Weight has remained constant. He sleeps fairly well and continues some activities.

The other gentleman continues with his work as a superintendent of his factory.

Will you walk across the room for us, please? Now will you walk backwards? You note the marked bending forward of the body and the rigid attitude and the continued movement of both hands.

Our next patient is age fifty-one, employed as a cashier; examined first on July 9, 1942. Family history is essentially negative. Her mother died of pneumonia at the age of forty-two; father died at the age of seventy-two, cause unknown. Five brothers and sisters are living and well. Husband died several years ago, probably brain tumor.

The patient has had no severe illness. Hysterectomy at age thirty-five, fibroid tumor.

Principal complaint was head tremor. Examination reveals this head tremor changeable in its rate and amplitude; smaller tremors of the hands, all of the type of release phenomena. The neurological examination showed all motor reflexes present and normal; pupils normal; eye-grounds normal; sensory field negative; blood Wassermann negative; spinal fluid was not studied; blood pressure continues about 140/85; urine normal; blood picture is normal. There are no gait abnormalities, and no Romberg; heart and lungs are normal; teeth required extraction.

You will note here a slight tremor of both hands and a facial tremor and once in a while we have rather severe release phenomena in which the head is moved around quite markedly. I believe it has not been noticed today.

Will you walk across the stage, please?

When she was first examined the pathology seemed to involve the head most particularly and my first thought was that it was a senile tremor, but I do not know whether it happened while she was in here or not but she would have those jerky movements of her head, marked contraction of the sternocleidomastoid. She is carrying on her work as a cashier and knows that conditions are not going to be favorable, never will be, but she is making the best of it, just like a man with a wooden leg who has to learn how to live with it. These folks unfortunately have to learn to live with their disease.

This next young woman has a different history. She is thirty-one, had whooping cough and measles in childhood; encephalitis, sleeping sickness, at the age of nine. Following this illness, a strabismus was noticed. Other symptoms developed slowly and she has been under our observation only since April of this year, when she complained of pains in her left arm, tremor, and general right arm rigidity, and excessive salivation.

On examination we find all motor reflexes present and exaggerated; pupils are normal; ocular motor nerves normal; muscular rigidity general, most marked on the right side. Here again there are areas of paresthesia and some areas of sensory dullness, especially the right arm, and to some extent the right side of the body. She has fixed facies, slight tremor of the tongue; urine normal; Wassermann negative. She maintains her body weight and can perform easy tasks at home. Her illness coming at the age of nine, she did not have the advantage of much formal education, so she has been obliged to carry on and do little simple things at home which she is able to do.

Will you walk across the floor once? You note a slight drooping of the left eyelid and continuous tremor of the arm, particularly the right arm, rigid attitude and fixed facies.

You always wonder what you can do for these folks. There is something to do after you have made a diagnosis. My surgeon friends say to me: "What's the use of being a neurologist? After you have made a diagnosis there isn't anything to do but give your patients a K. I." It is not altogether true. You can keep up their morale if you do nothing else. These folks should be taught to be doing something useful. For many years after a paralysis agitans begins to develop these folks can be useful citizens and keeping up their morale is very important.

As to medicines, of course, as you all know, the belladonna group still has its place. Hyoscine hydrobromide is used probably as much as anything else, and tincture of stramonium. I have found one of our pharmaceutical houses makes a tablet that is made up of mixed alkaloids of belladonna that has been very useful to me. You may have to use the bromides or sedatives of various kinds, and sometimes they have such terribly intractable headaches that you may have to resort even to an opiate to give them relief; you won't want to, but you may be obliged to.

One of my patients had a very troublesome diaphoresis. He complained dreadfully about sweating, great outbursts of perspiration. I remembered that sometimes the estrogens are used in preventing sweating in cases of severe burns to prevent loss of fluids through the skin and so I gave this man over a period of three or four weeks at weekly intervals, 2,000 units of an estrogen and for about four or five months he has had no sweating and does not complain.

I do think that one should not feel that there is nothing to do about these cases, because there is something to do and if you do nothing more than keep up their morale and keep them useful citizens as long as possible, that in itself, is something.

DIAGNOSTIC SURGICAL CLINIC

Surgical Diseases of the Colon and Terminal Ileum

By FRANK H. LAHEY, M. D.
Lahey Clinic, Boston, Massachusetts;
President, Inter-State Postgraduate Medical Association of North America

I would like to talk briefly, and largely without consideration of technical procedures, on four subjects, namely, polyposis of the colon, malignancy of the colon, ulcerative colitis and terminal ileitis. Since we have had a large experience with these conditions, deductions from our mistakes and success may be of diagnostic and therapeutic value.

Polyposis

First, I will speak briefly about polyposis. This disease involves eventually, we know, the entire bowel. We know its familial characteristics; therefore, we should remember that if one member of the family has it we must investigate every other member of the family and the children repeatedly. If we are going to control polyposis, we must catch it in the early stages. You can do very little with the condition when the polyps completely involve the rectum and sigmoid.

What can one do if polyposis is detected early? All the colon down to the sigmoid can be removed and a temporary ileostomy performed. The sigmoid can be implanted in the left lower quadrant so that a proctoscopic examination can be performed below and above. If the polyposis has not gone too far, the polyps above and below can be fulgurated out until the rectum and sigmoid are clean. The ileum can be anastomosed to the sigmoid, the polyps can be watched through a sigmoidoscope, and if they return, they can be fulgurated out.

If we accept the fact that practically all cases of polyposis ultimately become malignant, we can by the foregoing treatment successfully handle cases that hitherto were hopeless as to management. Several of our young patients have had the colon out down to low in the sigmoid, have had the polyps in their remaining sigmoid and rectum fulgurated, and are getting along well.

Carcinoma of the Colon

There is no reason why we should not improve upon our results, which are already good, in carcinoma of the colon. Here is a disease of low-grade malignancy, approachable surgically, with a high operability rate, a high five-year nonrecurrence rate, and usually giving early symptoms. In carcinoma of the lower colon, these symptoms include bleeding, discomfort, tenesmus, and change in bowel habits. In carcinoma of the upper colon, they include bleeding, mucus, change in bowel habits, anemia particularly in a lesion of the right colon, loss of weight and vague abdominal symptoms.

Dr. Neil W. Swinton, who in peacetime is in charge of proctology in the Clinic, reviewed the histories of 300 patients who were operated upon for carcinoma of the colon (100 carcinomas of the rectum, 100 carcinomas of the left colon, and 100 carcinomas of the right colon). He checked for answers to these questions: Was there alteration of bowel function? Were there abdominal cramps or pain, abnormal stools, blood or change of caliber of the stools. In the 300 cases, there was a negative answer to these questions in only 2.3 per cent of the cases. In other words, the diagnostic feature of malignancy of the colon and rectum was present in the history in 97.7 per cent of the cases.

With these facts in mind, we should, from the history, make the diagnosis oftener and earlier than we do. In practically every patient upon whom we operate, I can go back in the history and find a diagnostic point that should have prompted earlier and more complete investigation. Back of these diagnostic points are undoubtedly the basic promoting factors of carcinoma of the colon and rectum in so many cases—the polyps. We know that 75 per cent of carcinomas of the colon and rectum are in the descending colon, sigmoid, rectosigmoid or rectum. Sixty-five per cent of the polyps are in the same region. We see these polyps with malignant degeneration at the base, in the body and at the tip. Therefore, we know that we should pay more attention to the diagnosis and removal of polyps because they are so often the premalignant stage of carcinoma. The early colon polyps with a pedicle are easy to handle because one can, through a colotomy, remove a good area of mucosa about the base with the polyp. But with the broad-based polyp, each is an individual problem.

What should we do with polyps in the early stages if the pathologic report shows malignancy? In many instances, the epithelium covering a polyp is atypical. The diagnosis of malignancy in a polyp is not easy since it is difficult to differentiate atypical epithelium from true malignancy. If the pathologic report is malignancy in the body or tip of a polyp, should a radical operation be done? No, because it is not justifiable without malignancy of the base to put these patients through such a radical procedure. We take out a wide section of mucosa at the base and then watch the lesion by repeated postoperative roentgenograms. On the other hand, if there is clinical and macroscopic evidence of malignancy, plus mucosal ulceration at the base extending from the pedicle of the polyp over onto the mucosa, then radical removal should be performed. When the lesion is small, when the polyp has malignancy at the base and the ulceration has spread to the mucosa, do not let the family physician or the patient because of his desire for the natural method of defecation, persuade you to modify a procedure that will save the patient's life. The earlier the lesion, once malignancy is established, the more radical should be the surgical approach because of the increased chance for cure. Do not put radium on it or do local or segmental excisions. Do the radical abdominosacral removal and give the patient an artificial anus because he had better have an artificial anus with a cure than later a colostomy and recurrence.

What is the mortality of carcinoma of the colon and rectum today? We have operated upon 1,400 carcinomas of the colon and rectum, with a mortality of 10 per cent. In the last five years it has dropped to 5 per cent, and in the colon to 2.7 per cent with the Mikulicz procedures.

What is the operability? It has progressively risen to 82 per cent. In 82 per cent of the cases we can do a radical removal.

What is the nonrecurrence rate? In carcinoma of the colon and rectum combined, 50 per cent of patients who have had a radical operation are alive and well five years later. Therefore, we have here a great thing—a high operability, a relatively low mortality rate, and a high five-year nonrecurrence rate.

Can we do better? Yes, we can do better by watching the histories for indications of alteration in bowel function, pain, colic and abnormal stools, and making an earlier investigation and diagnosis. We can operate before the lesion becomes malignant by removing polyps.

What can be said about the removal of polyps? If there is unexplained blood from a higher level than the sigmoidoscope will show, that patient probably has a polyp unless he has a Meckel's diverticulum and aberrant gastric mucosa in it.

Should a polyp be explored? No, a polyp is difficult enough to find after roentgenologic examination has shown it. If the roentgenogram shows it once but not again, should one explore? No, I have done so unsuccessfully, and it is distressing. Do not explore until the roentgenogram repeatedly has demonstrated the presence and location of the polyp. A piece of feces can simulate a polyp. Never do it without a satisfactory bismuth contrast enema. Clean the patient out well, send him home with castor oil, and for colonic irrigation (mechanical); then give him another irrigation and do a contrast enema. If there is a polyp, then operate. If you are not sure, have him return in a few months, and if you can demonstrate the polyp again and again, you can then operate.

One other thing I would like to add is that I am absolutely certain, as I look back over the earlier years, that I sewed up patients with supposedly inoperable carcinoma of the colon who were operable. Why? Because I did not know enough about contact extension. I am certain that many patients with operable carcinomas of the colon are being sewed up as inoperable (and I do not mean this critically of others). Think how many carcinomas of the rectum are at the rectosigmoid where they literally lean against the back of the uterus. They involve the back of the uterus, and so because of contact extension they readily can be judged as inoperable by one inexperienced with carcinoma of the colon and rectum. We have now done abdominosacral removal of the rectum, uterus, tubes, ovary, and part of the vagina in over 48 cases. There is no reason why the uterus should not be removed, since in those cases the involvement is only contact extension.

Again, carcinoma of the sigmoid, because the sigmoid is mobile, gravitates to the top of the bladder, particularly in the male, and there is often direct extension from the sigmoid to the top of the bladder. The bladder can be freed and two-thirds of it removed, and it will stretch out into an adequate bladder. We have repeatedly excised the top of the bladder together with this contact extension in patients who were considered inoperable.

I have a woman vividly in mind who had been opened and closed because she had two loops of jejunum involved in contact extension of a carcinoma of the ascending colon. Dr. Cattell, at least five or six years ago, took out the two loops of jejunum which were involved by mere contact extensions; she had thirty-one lymph nodes with none involved, and she is alive and well.

We have seen these direct extensions in the right posterior gutter involving even the perirenal fat. We have repeatedly dissected the pelvis of the kidney because the perirenal fat was involved by posterior local extension. We have taken off the psoas or iliacus muscle because of posterior extension. These are the patients who I believe are not infrequently, and often were by us in the past, sewed up as inoperable when their extension was not to the lymphatic glands but was direct contact extension. I plead with you to have in mind that the operability of carcinoma of the rectum and colon can be greatly extended.

What should we do with the patients who have one or two metastases in the liver and a removable lesion? Take out the malignant colon lesion. I think there is something about the removal of the primary lesion that limits the rapidity of growth of the metastases in the liver. We have had patients live two or three years with metastases in the liver, after the removal of the local growth. It is well worth while doing those patients with only one or two small liver metastases, since they are more comfortable, have no pain, and their colostomy functions better.

What about a colostomy? Do not let anyone tell you that a colostomy is a living death. The public and unfortunately many family physicians have formed an erroneous opinion about the colostomy on the basis of the palliative procedure. Palliative colostomy is a living death, but it is not because of the colostomy. It is because of the irremovable malignant lesion, the pain, the blood, the pus and the mucus. However, a good colostomy after the lesion has been removed is not a living death. We have literally hundreds, 300 or 400 at least, of patients with colostomies who do everything and enjoy life.

What is the basis of the success of a colostomy? You must establish the patient's morale and educate him. Is there a trick colostomy which will have a sphincter action? No. Do not put the colostomy in the perineum because you then have to limit the extent of removal of the colon and rectum so that you can get the end to the perineum, you have to jeopardize the blood supply, and the only way you can see the colostomy is with a mirror. Put it up in the side with no twists and then teach the patient to constipate himself. The entire success of colostomy is constipation. Have the colostomy stick out above the level of the skin a finger's length so the edge will double back, and it will always be well above the skin edge. If it is below the skin level, it will close by contraction. It may close if it is above, but it is less likely to do so. Six months will be necessary for the scar tissue to contract so that it is about the size to admit the index finger, and for what is left of the terminal colon to learn to harbor and store feces. Teach the patient to irrigate himself once every two days or once every third day. He should devote that evening to his irrigation. Teach him to sit on a stool, using an apron or some other method, such as a tube, read a book, smoke and be comfortable. Teach him to put in at first a little soapsuds to soften up the inspissated button of feces that is the plug underneath the small colostomy. He should then put his finger in and break up the plug, then gently put the catheter in far enough, run in enough fluid and take time enough to expel it and get a good movement.

Some of these patients with cholostomies can stand over a toilet and literally empty their colostomies. However, most of them have to learn a constipating diet, the liberties they can take with it, and how to irrigate themselves. In general, we need to educate patients that a colostomy should in no way interfere with any activity.

An occasional patient gets a stricture. When a patient telephones me from a distance and says, "I cannot get my irrigation back; I put it in but it won't come back," I know what has happened. He has a keloid stricture about the colostomy. He needs to come back and have the keloid taken out and be instructed to put the finger past the middle knuckle through the colostomy preceding every irrigation.

We have here, I think, a field which is already a satisfactory one but one which can be made more so.

Ulcerative Colitis

We have had 400 patients with ulcerative colitis. It is in the beginning a mucosal disease. About half of the patients can be managed medically. What are the indications for ileostomy? If you wait until you, the family physician, and the patient's friends are all anxious to have the ileostomy done, you will be waiting until the patient is toxic, running a fever, with a distended belly, and then when you do the ileostomy, he will all too often die. He will die because his colon wall at that time is filled with virulent organisms, and its mesentery filled with infected thrombi. You should do it earlier, after the patient has had two acute attacks from which he has recovered. Do an ileostomy promptly after medical failures to relieve these acute attacks, certainly after massive hemorrhages (the patient can literally die in a week from massive hemorrhage in ulcerative colitis), in subacute perforations, in obstructions and when polyposis has occurred.

The so-called lead-pipe colon is the typical lesion of long standing ulcerative colitis. When an ileostomy is done, putting such a rigid colon at rest, unfortunately it cicatrizes and produces a stricture, resulting in an acute inflammatory reaction which may then indicate immediate colectomy.

We have to bear in mind that there is a stage in ulcerative colitis in which one can undertake surgical procedures other than ileostomy. Ulcerative colitis in 80 per cent of cases involves the rectum, but there are certain cases in which it is limited to the right colon. I have one at home now limited to the ascending and transverse colon only. In such a case you should remove the infected segment of bowel, put the ileum against the descending colon by the modified Mikulicz plan which I have described, just as you do in regional ileitis, and accept the possible opportunity to rid the patient of extension of the disease, because ultimately if left alone it will extend to the rectum. When, for instances, a lesion involves the other side of the colon, such as the rectum and the ascending colon, but not the transverse colon, an ileostomy can be avoided by taking out the rectum and the ascending colon and making a colostomy in the transverse colon proximal to the splenic flexure. In such a case, the colostomy is infinitely more desirable than the ultimate ileostomy which will have to be done if the lesion is left to extend and involve the whole colon. We have been, I believe, a little slow in our radical approach, particularly in partial involvement of the colon by ulcerative colitis. When such is the case, we should be quick, I believe, to excise that portion of the involved colon and save the remaining portion because if left alone, all of it ultimately will be involved and an ileostomy will become necessary. When, however, the entire colon is involved, as is so frequently the case in ulcerative colitis, then it is a different problem, and if colectomy must be done it will include abdominosacral removal of the rectum.

How many of these patients have we had to do a total colectomy on? We have done a total colectomy on 80 patients; we have done a partial on about 18 more. When should an ileostomy be done and when a total colectomy? An ileostomy should be done when you begin to wonder whether you should. If you delay beyond that, that is the time when you get into trouble. Do an ileostomy when there have been two acute fulminating attacks and in spite of medical measures, you have blood, pus, diarrhea and temperature.

When should you do a colectomy? If you have done an ileostomy and the patient goes on all right and gains 20 to 30 pounds, as these patients do, you do not need to do a partial colectomy, but if the patient has two acute fulminating attacks within the remaining colon with a discharge of blood and pus and with a fever, you had better do a colectomy before you get a perforation and before you are forced to do it in an acutely inflamed or perforated colon.

We have done a hundred ileostomies. In the first half the mortality was about 22 per cent because the family physicians and we waited too long. It is now about 5 per cent. When an ileostomy has been done and the colon has quieted down, can the fecal stream be put back? We have put it back in 15 cases. However, never put it back except when you can demonstrate by the proctoscope and sigmoidoscope that the rectal and sigmoid mucosa is intact; never except when you can demonstrate by roentgenogram that there are peristaltic flexible waves in the remaining colon and some haustral returns. If you put the fecal stream back with a narrowed, fibrosed and strictured rectum, it will blow open and a fatality will result.

Will you have to take the ileostomy down? We have, and we have had to do a colectomy in 5 of these patients, but if the patients understand the conditions, then we believe that to restore the fecal stream, if there is a good rectum, a good sigmoid and a flexible colon, is justifiable because ileostomy is such an undesirable type of thing.

Regional Ileitis

We have done resections of the terminal ileum, ascending colon and hepatic flexure in 75 cases. The history of regional ileitis is right lower quadrant pain, nausea, diarrhea, intermittent obstruction, weight loss, chronicity, often previous operation, appendectomy without relief, inanition, anemia, tenderness, palpable mass so often of the sausage-shaped type, and external or internal fistula into the sigmoid, vagina and perineum and through the abdominal wall—typical regional ileitis running from the ileocecal valve out.

What are some of the things we should know about regional ileitis?

First, it is undesirable to operate on acute regional ileitis during the inflammatory reaction stage when there is a lot of fibrin on the ileum and in the belly. Why? The virus, which it probably is, can be disseminated at that time. The patient is in poor condition for manipulation. The peritoneal cavity is not vaccinated against the lesion, and there is no harm in waiting three, four, five or six weeks. Don't operate in the acute stage if you can avoid it, and in many cases you can demonstrate them in the acute stage.

Secondly, we disapprove of all conservative lateral anastomoses and sidetracking procedures because of the number of fistulas we have had. We have 4 patients in the hospital now, 3 of whom have had fistulas into the colon as a result of delay. Do not operate in the acute stage, and when you do operate, take out the terminal ileum, the ascending colon and the hepatic flexure because all of these patients have glands in the mesentery of the right colon. Just as in ulcerative colitis, the inflammatory process often spreads over to the ileum, so in regional ileitis the inflammatory process often spreads over onto the ascending colon. Get out the terminal ileum well beyond the inflamed area, the ascending colon and the transverse colon, in order to get out the mass of inflamed glands in this right angle area. We take them out in two stages by the modified Mikulicz plan which I have described. I am quite certain that if you can get out all of the glands you at least increase the chances of nonrecurrence.

Thirdly, never fail to look the small intestinal tract over to be sure there is not a separate segment involved in regional ileitis.

The jejunum is peculiarly unlikely to be involved in regional ileitis but can be. We have never in our experience seen obstruction high in the jejunum, so I do not think many of the jejunal lesions will need removal. On the other hand, if a segmental lesion in the ileum exists, in addition to the one in the terminal ileum, take the segmental lesion in addition to the lesion at the terminal ileum.

In these 75 cases, has the disease recurred? Yes, in 5 patients who have been resected again. I believe those cases were early ones and that we did not know enough about the lesion then to do the radical removal of the ascending colon and the hepatic flexure together with the terminal ileum and the involved glands.

What is the mortality? In the 75 resections, 2 deaths have occurred, 1 due to an embolus and 1 to a leak of a primary anastomosis, which I did not do and would never have done. Technically, I think there is nothing more ideal for regional ileitis, for right-sided colon resection, than the so-called modified Mikulicz which I have described, whereby the terminal ileum, the ascending colon and hepatic flexure are removed, and then the ileum is placed beside the transverse colon, leaving, as you know, one segment of ileum which has been avascularized, sticking out longer than the colon, the so-called staggered loop, into which you can put a catheter right after the operation and get immediate decompression.

SUMMARY

I think in these four conditions we have excellent opportunities to make advancement:

1. In the polyposis cases, watch the cases in which we know the condition is familial, in order to discover it early and treat it radically but still preserve bowel control.

2. In the colonic carcinoma cases, remove the polyps early. Earlier diagnosis is possible from the history in which the diagnostic feature is present in 97.7 per cent of cases.

3. In ulcerative colitis, earlier ileostomy should be done before the patient is in too bad condition; earlier total colectomy after ileostomy has proved ineffective; earlier partial colectomy instead of an ileostomy when that part of the colon which is now involved can be preserved.

4. In regional ileitis, do not do a radical resection in the acute stage of the disease, remove all the infected ileum together with the ascending colon and hepatic flexure with all the right mesentery and its glands, and investigate other segments of the small intestine for possible separate segment lesions.

DIAGNOSTIC SURGICAL CLINIC

The Diagnosis and Surgical Treatment of Cancer of the Esophagus

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There has been a renewed interest on the part of the surgical profession in recent years in the treatment of cancer of the esophagus. I believe the reason for this is the general dissatisfaction with the hit-or-miss therapy usually accorded this disease throughout the world. Until recently the diagnosis of carcinoma of the thoracic esophagus carried with it a fatal prognosis and was followed by palliative treatment in the form of deep x-ray therapy with or without a permanent gastrostomy.

The final mortality with this treatment was close to 100 per cent. Inasmuch as this was the generally accepted method of handling this disease for many years, a large experience accumulated and it is now possible to scrutinize the effect of x-ray therapy especially from the standpoint of cure.

An enormous literature on the radiation treatment of cancer of the esophagus has accumulated throughout the world. Of the vast number of cases reported, approximately 20 are said to be cured. Careful study of the published results in these cases in the light of our present-day concept of cancer leaves many of them open to question because in a considerable percentage a positive biopsy was not reported and in the remainder the followup period was too short to warrant the contention that the disease was cured.

Except for an occasional recovery reported since Torek's original successful operation in 1913, the surgical treatment of cancer of the esophagus did not receive the impetus that it deserved until recent developments in the field of anesthesia and until the physiological changes in the circulatory and respiratory systems accompanying thoracic operations were more clearly understood.

With the latter, there has been marked progress in the field of thoracic surgery. Surgeons now enter the thoracic cavity with the same sureness as in performing a laparotomy. The impetus to attack cancer of the thoracic esophagus by radical surgical means has been strengthened by a gradually increasing knowledge of the subject of cancer in general and by a clearer understanding of the necessity of radically removing a cancer-bearing focus with all the lymphatic tributaries draining the particular site.

From a pathological standpoint, it is important to emphasize that cancer of the esophagus may remain a local disease for a considerable period of time. I should like to stress the fact that the life history of this disease in relation to the findings at operation is vastly different from the picture seen in the terminal stages on the autopsy table.

Although extensions to the regional lymph nodes may occur late, the tumor itself may become locally inoperable because of fixation to vital structures, such as the aorta, bronchus or vertebral column.

It must be remembered that the length of history of dysphagia does not picture the extent or size of the tumor. Dysphagia, the outstanding symptom of cancer of the esophagus, does not occur until gross involvement of the esophageal lumen takes place. The tumor may grow outwardly rather than toward the lumen of the organ and may reach an inoperable stage before persistent dysphagia becomes a prominent symptom.

A few years ago I was under the impression that all patients with a short history could probably be classified as operable, but following a rather extensive experience with this disease since then I have reached the opinion that too great stress should not be put on the duration of the history in determining operability. I have seen a patient with a history of a year's duration in whom a radical resection was carried out and on the other hand I have noted three or four other patients with short histories varying from one to three months in whom exploration revealed inoperable tumors.

In determining operability from the pathological standpoint, it must be emphasized that the location of the tumor is of great importance. Experience indicates that those tumors located in the middle third of the organ between the arch of the aorta and the left main bronchus quickly become inoperable because of early fixation to one or the other structure. Tumors located in the superior mediastinum just above the arch of the aorta likewise become inoperable early in the course of the disease because these are prone to involve the left recurrent nerve and seem to spread more quickly to the nodes at the base of the neck.

Fixation to the right mediastinal pleura does not by itself render the tumor inoperable. On four occasions I have been able to excise a portion of the right mediastinal pleura with the growth and successfully bring the patients through the operative ordeal in spite of a bilateral pneumothorax on the operating table.

Cancers of the middle third of the esophagus, that portion situated from the upper surface of the aortic arch down to a point about 35 cm. from the upper incisor teeth, metastasize to the mediastinal lymph nodes, and late in the disease to the supraclavicular nodes on the left side.

Occasionally retrograde metastasis may take place downward below the diaphragm to involve the nodes in the paracardial region and in the gastrohepatic omentum.

Cancers located in the distal third of the organ, the most common site, that extending from the lower part of the middle third to the cardiac end of the stomach, usually spread to the regional nodes in the mediastinum. Not infrequently there is extension below the diaphragm to the paracardial and gastrohepatic nodes. Involvement of the liver is, in my experience, a most unusual route of extension.

It is important to emphasize the implications of the nature of the histological examination of the biopsy specimen obtained by esophagoscopy. The finding of squamous cell characteristics in the tumor indicates origin of the growth from the esophageal mucosa. If adenocarcinoma is found on histological examination, there is definite evidence that the tumor arises from mucosa of the cardia or the upper portion of the stomach. I have heard clinicians and pathologists state that adenocarcinoma of the distal esophagus often originates from misplaced gastric mucosa in the esophageal wall. I believe this to be grossly inaccurate. A fairly large operative experience indicates that every instance of adenocarcinoma of the distal esophagus has demonstrated that the tumor originated in the cardia or upper portion of the stomach.

The lymph-node spread from adenocarcinoma of the cardia is mainly below the diaphragm, and in the majority of instances the extension is in the direction of the paracardial and gastrohepatic nodes. Occasionally the peripancreatic nodes may become involved. Extension to the portal system with metastatic involvement of the liver may occur early in the disease. In a few instances I have noted extensive node involvement in the mediastinum above the diaphragm with minimal involvement below the diaphragm. This clear distinction of the difference of lymphatic spread of squamous cell carcinoma and adenocarcinoma is the reason for my suggestion that every case of adenocarcinoma of the lower esophagus should be explored through an abdominal incision in order to determine operability.

Cancer of the esophagus occurs most frequently in the sixth and seventh decades of life. I have seen two patients in the fourth and many in the fifth. The disease apparently is more common in men than in women. There is nothing in any of the histories that I have elicited which would indicate some predisposing cause such as chronic irritation. Without exception, every patient has presented the one cardinal symptom of dysphagia, that is, interference with the normal act of swallowing. Most often this difficulty is concerned with the swallowing of solid food. The patient usually states that swallowed food seems to stop at a point in the esophagus which the patient indicates by pointing to a portion of the sternum. Most of the patients have been quite accurate in localizing the site of obstruction, when this was compared with the x-ray film and the findings at operation.

Pain on swallowing has not been a prominent symptom in the cases I have seen. In almost every instance dysphagia has been progressive, with gradual elimination of solid food from the diet. Most of the patients were able to subsist only on liquids at the time of the first examination.

The extent of loss of weight has been of considerable interest in determining the duration of the disease and arriving at an estimation of operability. My experience to date would seem to indicate that too much reliance should not be placed upon weight loss as an indication of the extent of the disease. The tumor which early impinges upon the lumen will produce a rapid weight loss early in the course of the disease. I have seen one patient who was known to have cancer of the esophagus for one year and yet there had been no weight loss whatever. By the same token, the extent of weight loss has not proved to be of any significance in arriving at an opinion concerning operability.

The symptom of persistent backache or back pain is of serious importance. It usually indicates extension of the growth beyond the wall of the organ, with fixation to the aorta or vertebral column. With adenocarcinoma of the cardia, backache may mean pancreatic extension. However, the symptom of backache does not necessarily indicate inoperability, because it may be caused by inflammatory reaction in the periesophageal tissues.

Every patient with carcinoma of the esophagus should be carefully examined for the presence or absence of an enlarged supraclavicular lymph node, so-called node of Virchow. The finding of a firm supraclavicular node or any other node in the neck demands biopsy before attempting surgical excision of the esophageal tumor. If metastasis is demonstrated on histological examination the case should be classified as inoperable.

It is needless to stress the fact that every patient who complains of persistent dysphagia should be subjected to careful roentgen examination of the esophagus and upper stomach. A negative examination does not exclude the presence of a neoplasm of the organ. If the symptom of dysphagia persists in spite of symptomatic treatment, examination should be repeated after three or four weeks.

I am going to show you some of the x-ray films in representative cases with the pathological specimens of the excised tumor. This is the x-ray film of a carcinoma of the middle third of the organ. (Slide) That is the gross specimen showing the growth around the periphery of the organ rather than in the lumen. Here is another in the middle third of the esophagus with almost complete obstruction.

(Slide) This shows a constricting type of tumor with a large amount of fibrosis in the wall.

(Slide) Here is another in the middle third of the organ.

(Slide) This is another variety of carcinoma of the esophagus, a shallow ulcer with overhanging edges, similar to the epithelioma one sees on the skin.

(Slide) There is another defect in the esophageal wall on roentgen examination, and this is the gross specimen showing the marked constriction in the lumen. Early weight loss is a prominent symptom in these cases. Here is another defect of the esophageal wall without complete obliteration of the lumen.

This is the tumor which did not completely obliterate the lumen.

Here is a tiny defect in the esophageal wall which was almost missed by the roentgenologist. He gave the patient the heavier mixture of barium and this shows the projecting small tumor in the wall.

This is a very early carcinoma of the esophagus. This patient is alive and well after seven and a half years after.

Here is another variety without complete obliteration of the lumen.

This is a colored photograph showing the tumor.

This is a carcinoma at the lower end of the esophagus with invasion of only the distal two inches.

There is another view showing the constriction of the lumen.

Finally, the gross specimen, showing the projecting polypoid tumor.

This is an adenocarcinoma arising from the greater curvature of the stomach and extending up the esophagus. Notice the finger-tipping, the irregularity of the greater curvature in the silent area of the stomach.

This is an adenocarcinoma of the cardia with extension up the esophagus showing the constriction.

This is the gross specimen.

This is another adenocarcinoma of the cardia with extension up the lower end of the esophagus.

This is the gross specimen.

The finding of an irregularity or filling defect or an obstruction on fluoroscopic and x-ray examination calls for esophagoscopy by a competent operator. There seems to be an idea in the medical profession that esophagoscopy is a dangerous procedure. There is little argument with the fact that in the hands of an inexperienced operator this is quite true. However, with a disease such as the one under consideration it is important to verify by direct visualization and by biopsy examination the presence or absence of a carcinoma before attempting radical surgical extirpation. Therefore, the alleged danger of the esophagoscopic examination itself has little significance when compared with the necessity of establishing a positive diagnosis.

Esophagoscopy has a twofold purpose. In the first place, the gross appearance of the tumor is noted and a biopsy specimen is taken to determine the histological characteristics. Secondly, the esophagoscopist notes the distance of the growth from the upper incisor teeth. This in conjunction with the roentgen film aids the surgeon in deciding the type of operation he will undertake. As stated before (and it is necessary to emphasize this point) demonstration of an adenocarcinoma in the biopsy specimen calls for preliminary abdominal exploration to determine operability, because of the direction of lymphatic spread that occurs with this tumor. With tumors located in the middle third of the organ one type of operation is indicated, while with tumors in the distal third beyond 35 or 36 cm. from the incisor teeth, another operative procedure is carried out.

The decision to undertake radical surgical extirpation of a cancer of the esophagus will rest on a number of factors. The age and general condition of the patient are of considerable importance. It would be poor judgment, in my opinion, to subject a patient in his seventies with a poor heart, arteriosclerosis and poorly functioning kidneys to the hazards of so extensive a procedure. On the other hand, I have successfully carried out a one-stage resection on a man of seventy-three years. Each case must be judged on its own merits, and it is important to have the help of a competent physician to aid the surgeon in estimating a patient's physical and physiological status.

Demonstration of metastatic involvement of the supraclavicular nodes indicates inoperability. Tumors located between the arch of the aorta and the left main bronches present a special problem. Fixation to either or both structures takes place early and the technical difficulties resulting therefrom may be formidable. The outlook from the standpoint of cure should the patient survive the operation is not favorable.

The duration of the history of dysphagia is of some importance in determining operability, but I do not at the present time consider it as important as I formerly did. Intra-luminal extension of the tumor and a degree of fibrotic constriction of the circumference of the organ are important pathological reasons for the severity of the symptoms. These must be carefully appraised.

Once the decision to undertake surgical treatment is made, the patient's general condition and his physical reserve after thorough preoperative preparation must be carefully appraised. It is important to pay special attention to oral hygiene. Great weight loss may be due primarily to extreme dehydration. The daily intravenous injection of 1,000 to 2,000 cc. of 5 per cent glucose in saline over a week's period often results in astonishing improvements of the patient. In a recent case the patient gained 10 pounds in seven days. Unless complete obstruction is present the patient should be encouraged to take large quantities of high caloric liquids containing the necessary vitamins and minerals.

In many instances parenteral administration of vitamins will be necessary.

It will be found that taking one or two weeks to improve the patient's general condition is time very well spent.

The question of preliminary gastrostomy deserves special attention. Until recently I was of the opinion that most patients required a preliminary gastrostomy to aid in restoring fluids, proteins and so forth. At the present time I have the opposite viewpoint and believe that except for the rare exception most patients can be gotten into satisfactory condition by the methods above enumerated. To aid in restoring lost proteins I have in the last few patients given amino-acids as advocated by Elman, and plasma.

Should alimentary feeding be necessary, jejunostomy instead of gastrostomy should be performed in the lower part of the abdomen.

In most instances it will be necessary to administer a blood transfusion before operation. A transfusion during operation is obligatory. A third transfusion during the early period of convalescence is also most desirable.

In my experience, the combination of avertin and cyclopropane has been most satisfactory.

Complete collapse of the lung should not be permitted to take place at any time and the anesthetist should have complete control of the situation. It is important to secure the services of a competent anesthetist because the incidence of postoperative pulmonary complications depends in large measure on the skill of the administration of the anesthetic agent.

The ideal to be sought after in the surgical treatment of cancer of the esophagus is radical resection of the cancer-bearing portion of the organ with all the associated lymph nodes and reestablishment of esophago-gastric continuity. With tumors located in the middle third of the organ it was impossible until recently to attain this ideal to its fullest extent. Although the Torek operation stood the test of time and experience I always believed that it was a makeshift procedure leaving the patient with an artificial esophagus, which was far from desirable from all standpoints. Because of this I am proposing a new operation which will eliminate these objections and I am reporting it today for the first time.

(Slide) This is the original Torek operation. The incision is made in the left chest, trans-thoracically.

This shows the left chest cavity with the mediastinal pleura incised. The esophagus is taken out of its bed. The tumor-bearing portion is excised with all the adjacent areolar tissues of the mediastinum. A modification, reported some years ago, was the preservation of the distal portion of the esophagus attached to the stomach in order to preserve the cardiac sphincter mechanism. This is brought out through an incision in the abdomen. The infra-aortic portion of the esophagus is brought up to lie above the arch of the aorta and placed in the root of the neck, the mediastinum being left open, the distal part inverted through the diaphragm into the abdomen. This shows the closure of the chest wall. The patient is turned over on his back, an incision is made along the left sterno mastoid muscle and the esophagus is drawn out through the incision. A subcutaneous channel is made from the lower part of the cervical incision down to the second rib on the left side and the esophagus drawn through this channel to form an esophagostomy opening over the second rib on the left side.

The new procedure is the same approach through the left chest with the splitting of three ribs above and two ribs below; the same incision in the mediastinal pleura. The tumor is located just below or behind the arch of the aorta.

This shows the amputation of the esophagus at the cardiac end of the stomach, withdrawal of the tumor upwards to lie above the arch of the aorta, amputation of the growth above the arch of the aorta, with preservation of the blood supply of the remaining stump.

This shows the amputated organ.

This shows the precise ligation of the blood vessels along the greater and lesser curvatures of the stomach in order to preserve the blood supply of the organ when it is transferred to the apex of the chest.

This shows the stomach drawn to the top of the chest over the aortic arch, and an anastomosis being effected between the stump of the esophagus and the stomach.

This is the completed operation.

I would now like to show examples of these various operative procedures.

This is a patient alive seven years with the upper esophagostomy opening and the lower gastrostomy opening. This is the tube mechanism replacing the removed esophagus. This is after a partial ante-thoracic esophagoplasty with the tube of skin ending at the lower sternum and the remaining distance taken up by a large rubber tube.

This is another patient with preservation of the distal part of the esophagus near the cardia and the upper esophagostomy opening.

This is the operative wound, and this is the rubber esophagus in place.

This is the roentgenogram of the patient upon whom the new operation was performed for a carcinoma situated just at the arch of the aorta, and this is the postoperative film showing the stomach up at the apex of the chest.

With tumors located in the distal third of the organ or in the cardiac end of the stomach the operation of choice is a left transthoracic, transdiaphragmatic resection of the lower esophagus and upper stomach, mobilization of the proximal two-thirds of the stomach and a suture anastomosis between the end of the severed esophagus and the upper end of the stomach.

These slides indicate the operation for tumors at the lower end of the esophagus and upper end of the stomach, left transthoracic approach. Incision of the mediastinal pleura and diaphragm. Mobilization of the stomach by ligation of the blood supply along both curvatures, amputation of the stomach and the esophagus above the tumor; closure of the stomach; drawing of the stomach up to the stump of the esophagus, and the performance of a careful two-layer suture anastomosis, using interrupted silk. The stomach is then drawn upward and telescoped over the esophagus and anchored to the mediastinal pleura and diaphragm and there is the completed procedure.

This is one patient who had this operation.

(Slide) The operative wound; under-water drainage scar.

This is a postoperative roentgen film showing the transplanted stomach.

This is the same patient.

This is another patient who had a transplanted stomach.

The thoracic wound.

(Slide) This is another film of a third patient with a transplanted stomach.

The general problem of the surgical treatment of cancer of the esophagus may be reduced to cold figures. The average patient with a cancer of the middle third of the organ stands a 60 per cent chance of surviving the operation and a 30 per cent chance of living more than one year. With squamous cell cancer of the distal third of the organ the patient has an approximate 70 per cent chance of surviving the operation and almost 100 per cent probability of living more than two years.

In the group of adenocarcinoma of the cardia the probability of operability is only about 37 per cent. There is a 60 per cent chance of operative survival and an 80 per cent probability of living more than one year.

Such is the present status of the operative treatment of this disease. The figures presented indicate a great advance over those given heretofore for the treatment of this disease by other methods. However, there is much to be desired. I am firmly convinced that the operability percentage, the mortality rate and the postoperative survival period will be markedly improved when more early cases are referred for operation and with increasing experience in the operative management.

DIAGNOSTIC SURGICAL CLINIC

Carcinoma of the Colon

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I have the Chair's permission to interpolate one remark here that I was fearful might not be in order, and that is to say that I think it is a very splendid decision made by the officers of this Association that this meeting should be held at this time. With the increasing difficulties in transportational facilities and with the increasing duties that the civilian physicians find themselves confronted with, I think that we have gone a little too far in the abatement of medical meetings, and I am confident that meetings of this kind which are in effect postgraduate schools are not only of the greatest value but that it is imperative that we continue to have this kind of association.

I know of no profession or group whose sacrifices in this war have paralleled the sacrifices of the medical profession, and with some 50,000 men now with the Colors in the Army and the Navy, the enormous burden of work which has been put on the medical schools with their accelerated curriculum programs and with the amount of work that they are doing in research, with the amount of time that their faculties are giving to aid the Army and the Navy in other directions, I think it is particularly fitting that we stop for a moment and consider that after all the profession owes to itself and to the public as well some recognition of the fact that medical education has to continue and that medical practice on a high standard has to continue, and this kind of a meeting is one answer to many of the complexities that have forced us to abandon gathering together for the last two years.

Since I have been on duty in the Army for a year and a half I have not had the opportunity to appear on programs of this kind often, and when I was invited to give a diagnostic clinic on cancer of the colon I visualized a number of patients who were in various stages of the disease before they were operated on and some after successful operation, with their accompanying histories and x-ray pictures; but I find that there are no patients, and having no slides and no paper, I find myself as barren of equipment as a traveling salesman who has forgotten his samples. However, I would count that day lost that I did not make a few remarks on cancer of the colon, given the opportunity. With your indulgence I would like to discuss some of the problems which are familiar to you and which probably will stand some repetition.

I think if we visualize the colon—and I mean the colon and the rectum as a bi-functional organ—it will assist us in diagnostic procedures as well as in the selection of the proper operative procedures to cure cancer of the colon and the rectum, and I say operative procedures advisedly because I know of no cancers of the colon that have been cured by any other method and I could almost add the cancers of the rectum to that group.

If we remember that the colon is developed not all in one piece but that beyond, to the left of the middle of the transverse colon, the rectum and that part of the colon lying there are developed from the hindgut and that the rest of the colon is developed with the small gut around to the ampulla of Vater, the midgut, we immediately recognize the functional differences in the two halves and thereby I think have a clue to some of the problems of diagnosis and some of the reasons for choice of different types of operation in the two halves of the bowel.

I think we should remember that cancer of the colon is probably the one place where we can prove with a considerable certainty that a large group, perhaps a large majority, of the tumors develop, as Dr. Lahey told you this morning, from polyps. I believe that that work has been conclusively and successfully carried on not only in Europe but in this country, and from my own experience I am convinced that a great many, perhaps most, cancers of the lower bowel do have their initial development in polyps. More and more polyps are found as we examine more and more colons at operation and at autopsy, and we have been able many, many times to trace the genesis of a cancer from a polyp, whether the thing initially began in its tip or began further down and extended into the base.

Symptomatically I would like to divide the cancers of the right and the left colon very sharply. It is true that many of the symptoms are the same in both halves of the colon, but an alert taker of histories will be able to localize carcinomas of the colon very largely on the story as given by the individual suffering from the malignancy.

I like to divide cancer of the right colon into three different groups: First, the anemic group; second, the dyspeptic group; third, the accidentally discovered group.

All of the symptoms of rightsided colonic cancer revolve around physiologic disturbances. The right half of the colon is the absorbing portion of the colon; the left half of the colon is merely a storehouse. Less than 10 per cent of the liquids, the content of the large bowel, are absorbed in this portion.

The anemic group of carcinomas of the colon probably comprise a third of the whole. That is a very distinctive group because these individuals do not know that they have carcinoma of the colon generally. They do not, in my experience, have pain or very much discomfort. They notice that they tire easily; they notice that they are run down, that they are unable to carry on their accustomed duties and that neighbors and friends not infrequently say to them that they look anemic and pale and that their mucous membranes are washed out.

Those people do not necessarily have a tumor which is palpable, and the only way that one in many instances can make a diagnosis is by careful radiologic examination.

Occasionally a hematologist can give you an accurate clue to the picture by directing your attention to the fact that this is not a primary anemia but a secondary anemia. These people present the picture very frequently seen in the old-fashioned type of pernicious anemia, and save only for women with bleeding fibroids I know of no more likely diagnosis in a profoundly anemic individual who has a secondary anemia and has not vomited blood than carcinoma of the right half of the colon.

The second group, or the dyspeptic group, in the earlier part of their disease suffer from vague indescribable dyspepsia which they try to get rid of in various ways by the use of various medicaments, and when self-treatment fails not infrequently are prescribed pepsin and various other things by someone who has not examined them. This group too will only be detected by careful roentgenological examination.

The third group, the accidentally discovered group, increases in direct ratio to examinations made by physicians or by the patient's own self, following some visit to a meeting of the American Society for the Prevention of Cancer. I think about ten per cent of all cancers of the right colon are discovered accidentally as a lump either on a routine physical examination or the patient without suspecting that he or she has any ailment, feeling the abdomen, discovers something unusual there.

On the left side of the colon things are entirely different, the pathology is different, the symptomatology is different, and the type of operation called for is entirely different. Symptoms of leftsided colonic cancer revolve around obstruction. They are symptoms of obstruction. Carcinomas of the left side of the colon almost invariably produce obstruction in some degree, and I repeat that, that there is obstruction in some degree in practically every carcinoma of the left half of the colon. Even after careful preoperative preparation one may see telangiectasis and thickening and a little edema proximal to the obstruction which is closing up the bowel.

I do not mean that one should wait until obstruction becomes so pronounced that one suspects carcinoma of the left half of the colon, but I do assert that obstruction in some degree is present in practically every carcinoma of the left half of the colon unless it is accidentally discovered or there is some reason for doing a barium enema which shows it up.

The early symptoms of cancer of the colon as so graphically described by Dr. Lahey this morning, of pain and discomfort, change of bowel habit, are very important. I think the most important is change of bowel habit. It is very difficult to elicit a history of change of bowel habit in many individuals, and yet if one keeps at it, if one suspects a carcinoma of the colon or suspects some colonic ailment, change of bowel habit is very likely to be elicited on close questioning. Some short periods of diarrhea alternating with short periods of constipation which call for a laxative usually mild in character should put one on guard that there is an organic lesion in the lower bowel and should call for a careful examination at the hands of a roentgenologist.

Let me say that I am sure that all cancers of the rectum can be diagnosed without an x-ray, and I think they should be diagnosed without an x-ray. All cancers of the colon can be diagnosed with an x-ray and usually that is the only way they can be accurately diagnosed and localized.

I take it that there will be small disagreement with the thesis that every patient with such complaint as we have been discussing should in the course of a routine physical examination have a rectal examination, a digital examination, which will show the presence of 90 per cent of the cancers. Perhaps I am a little high, let's say 75 per cent of the cancers. Some at the rectosigmoid juncture will not be felt by the examining finger even if the individual strains down during the course of the examination. On the other hand, every cancer of the rectum—and I mean cancer of the rectosigmoid also—can be easily visualized by a proctoscopic examination, and that, as Dr. Garlock said of esophagoscopic examination, is a difficult problem for an unskilled person, perhaps, but a relatively simple procedure for anyone even with a cursory training in proctology.

I mention this because I think that if there is a question of cancer of the colon, before any diagnostic procedures are indulged in which require the introduction into the bowel of an opaque medium, a proctoscopic examination should be done first. There is no question of doing a proctoscopic examination after you have done the barium enema. It should be done first, and for two very good reasons: (1) You find a great many cancers of the rectosigmoid that way when you are looking for a cancer of the colon; (2) you should know that there is no obstructing lesion at the rectosigmoid before you introduce a barium medium by bowel for an x-ray of the colon, and let me say right here that you should never give one by mouth. It is not only useless, but it is dangerous. You cannot get a good picture of the colon for diagnostic purposes by the introduction of the opaque medium by mouth, and not once but many times all surgeons of wide experience have been called upon to decompress individuals in whom an acute intestinal obstruction has been produced by the opaque media being given to an obstruction carcinoma of the left colon.

I should like to be very emphatic about that. One should never give a barium meal by mouth to diagnose carcinoma of the large bowel for those reasons that I have outlined.

Having properly diagnosed the individual, the choice of operative procedure, I think, is relatively narrowed down to a few procedures. I believe that all surgical procedures should follow a period of careful preoperative preparation in the hospital. I do not send my patients out; I keep them in the hospital for a week and see that they are properly decompressed so that I know they are properly decompressed and do not take their word for it. They are properly decompressed by giving an initial purgative and by subsequently following that up with many, many enemas until the bowel is washed clean.

I have no enthusiasm for the new sulfa cure-all drugs in the preoperative preparation of colonic cases for operation. I have had no experience with them, but I have noted very definitely that they change as the seasons of the year change and that with each new one offered on the market for this particular reason some of the ill effects of the old ones are finally brought to light. I think we have been doing very well without the sulfa drugs in the preparation of patients for operation, and if you want to give them after operation that is an entirely different thing, or if you want to elevate the blood level before operation, I have no quarrel with it, but the sterilization of the gastrointestinal tract by the oral administration of these drugs leaves me a little cold.

I have no enthusiasm, either, for the use of intraperitoneal vaccines. I share a certain responsibility for having supported that procedure vigorously some ten or fifteen years ago, but I found the error of my way and I have abandoned the procedure.

In choosing an operation for the right half of the colon I should say that one may not infrequently with success employ the one-stage operation, more frequently I believe one will be gratified to employ the two-stage operation because there does seem to be considerable danger of sepsis (not from leakage of anastomosis—I call that bad surgery; an anastomosis should not leak if you do it right); but from mobilization of the growth. Anyone who utilizes a procedure such as the Mikulicz or any other procedure which mobilizes an infected growth right then runs his chance of the lethal peritonitis which is the cause of mortality in all of these cases, practically. If you get by that particular stage you might just as well go ahead and do the anastomosis at the same time, in my judgment. One can do an anastomosis, though, and bypass the growth which is fixed to the lateral wall, fixed posteriorly in many cases, and those are the cases that Dr. Lahey was talking about that are now shut up and called inoperable. Those cases will give you a high mortality if you don't bypass the growth and reduce the infection around the growth, because it has been shown many, many times that the ulceration and obstruction increase the permeability of the bowel wall and thereby permit the organisms which are normally present to migrate, and in the very procedure of mobilization you give the individual a peritonitis.

I prefer, unless the growth is very early, very movable, very easily resected, to do an end-to-side or side-to-side aseptic anastomosis between the terminal ileum and the transverse colon and two or three weeks later go in and do the resection of the right colon.

If one wants to do a primary resection and anastomosis I think his criteria should be about those I have outlined: freely movable growth early; no obstruction; no evidence of fixation, but I do not get to operate on many of that kind. The ones that I see are generally fixed and they are generally not too early.

In the left half of the colon I believe that all of us like to do a primary resection and get rid of the growth. Then the debate comes as to the advisability of a primary anastomosis or leaving it open. I personally do not like to do the primary anastomosis in the left colon. I think you can get more bowel out, I think you can do a wider, a more thorough resection of the colon by leaving the clamp on, doing an obstructive resection, and if you supplement that resection with a cecostomy which is a relatively simple matter and requires only a few moments and a small tube, I believe that you will find it one of the most satisfactory of all types of procedures for cancer of this particular location.

There are many modifications and variations of these particular types of operation which have to be applied to the individual case, for all of these cases should be accepted on their merit and should be individualized and not fitted to an operation. The operation must be fitted to the patient, of course.

The essence of an operation is a widespread resection of the bowel and the tissues in the immediate vicinity of the growth. The planned dissection can be done in dealing with cancers of the large bowel, and it is an essential part of the whole procedure.

I think that the encouraging part of surgery of the large bowel is the good prognosis which follows radical resection, and all surgeons who have had wide experience in this field have extended their field of operability, have increased their number of resections, and have lowered their mortality figures. My mortality figures range around 6 per cent for the rectum following the one-stage combined abdomino-perineal resection, and about 8 per cent for the colon. I have not been able to reduce them below that for either location. As for the resectability figures, my resectability figures the last time I looked them up were 72 per cent resection, not in the whole series but resection in the belief that I was doing a radical operation for cure. I believe, too, that an individual is more comfortable to die from liver metastasis than from obstruction from a cancer in the bowel, and I believe that many resections are justifiable for that very reason. However, I don't think that that particular group counts in the resections for cancer where we are looking forward to a more satisfactory prognosis.

I think, therefore, that we can say that cancer of the lower part of the gastrointestinal tract gives an infinitely better prognosis than cancer of the stomach, for instance, and I think that with the improved operative technic, better preoperative preparation, better selection of cases for operation, we can give these people an infinitely superior outlook.

Precautions against the Introduction of Tropical Diseases into the United States

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INTRODUCTION

Probably at no time in the history of the world has there been such universal interest in tropical diseases as we find at the present moment. In the past, the majority of medical men in the United States, particularly those in the northern states, have shown little concern about tropical diseases. More recently, as we stood on the brink of war and after our entry into this global conflict, this lack of attention on the part of the many has turned to real concern regarding the possibilities of the introduction of tropical diseases into this country which might result in epidemics of major proportions. Therefore, it is timely that the subject of tropical diseases be discussed anew. We need to carefully evaluate the influence that tropical diseases may have on the outcome of the present world struggle, and on the future development of international relations and commerce in the period immediately following the cessation of hostilities, and for many years to come. As we stand at this cross-roads we need to consider the problem with an open mind, and decide carefully on the attitude we are to take toward tropical diseases, for they have in the past, and may in the future determine the progress of civilization in our nation and other nations of the world.

In order to expedite this discussion I wish to define and limit the term "tropical diseases." The tropics are defined geographically as including the equatorial area between the parallels at 23 degrees and 27 minutes North and South latitudes, or that they are located between the Tropic of Cancer in the Northern Hemisphere and the Tropic of Capricorn in the Southern Hemisphere. From the standpoint of the temperature, the tropics are the areas within the 68 degree F. isotherms; the regions in which the mean monthly temperature of the coldest month is never under 68 degrees F. Likewise, we may define the sub-tropics as areas adjoining the tropics in which the mean monthly temperature for the whole year is never under 68 degrees F. There is considerable divergence of these isothermal lines from that of the geographic parallels limiting tropical areas, this being due to the influence of water currents, air currents, and the physical variation in the earth's surface evidenced by mountains, valleys, islands, and bodies of water. Within the geographical tropics we may have climatic conditions in the areas of high altitude that simulate those of the temperate zones. Large land areas may encompass teeming jungles or swamps, or the opposite extreme of a desert, depending upon the prevailing precipitation. Actually it is not possible to accurately define the tropics in a geographical sense.

For convenience, the term "tropical diseases" may also be limited in its scope. If we limit the subject narrowly to include those diseases that are found only in the tropics, we would confine it to one or a very few diseases, the most notable of which is African sleeping sickness. In this discussion I wish to include those infectious diseases which are especially prevalent in warm climates, and to pay particular attention to those that could possibly be introduced into the United States. In this group are diseases that can be and are successfully propagated in cooler climates, but are not found in these areas to any great extent because of certain limiting factors. The amount of tropical diseases in a population group or area depends primarily upon the level of personal and group hygiene, the presence of proper intermediate hosts, such as Arthropods, and the climate. The prevalence of certain diseases in warmer climates is influenced markedly by the lower standards of sanitation and hygiene found to exist among natives of these areas, and these appear in a vicious cycle associated with adverse economic and social conditions. Certain of these diseases can be propagated and disseminated only through the aid of a particular intermediate host, frequently belonging to the Arthropod group, and these in turn are found to be more abundant in warmer climates. The climate of the tropics, because of the favorable conditions of temperature and moisture that usually prevail, offers an ideal situation for the rapid, uninterrupted propagation of pathogenic organisms, particularly those that inhabit the intestinal tract of man, those that require an Arthropod intermediate host, or those that must spend a period of development or incubation outside the human host. Other factors, such as the excessive demands of the climate on the individual and malnutrition, may lower the resistance of the person to attack by disease organisms and may lead to a higher incidence of a disease and/or a greater severity in its clinical manifestation. As has been pointed out by Dr. W. A. Sawyer, the term "tropical" refers to a type of environment, and that a tropical disease is in reality any disease as it behaves in a tropical environment. He also pointed out that it is necessary to stress environment and epidemiology, as influenced by environment, if prevention is to gain its rightful place in the tropics, where so many of the diseases are essentially preventable.

The speaker makes no claim to originality for much of the subject material presented herewith, for it has been obtained from many sources in the literature of Parasitology and Tropical Medicine, from conversations with persons who have been in recent close contact with the problems to be discussed, and from limited personal experience in tropical areas of both Eastern and Western Hemispheres.

The Introduction of Tropical Diseases into the United States in the Past

We have within the continental limits of the United States today certain tropical diseases, and others have been present in the past, but have since apparently disappeared. Among these may be mentioned the dysenteries (amebic and bacillary), yaws, leprosy, yellow fever, cholera, dengue or "breakbone fever," plague, relapsing fever, epidemic and endemic typhus, Rocky Mountain spotted fever, "Q fever," malaria, Bancroft's filariasis, dracontiasis, hookworm disease, and perhaps other helminthic diseases. Dr. H. Harold Scott in his recent volumes "A History of Tropical Medicine," and Dr. E. C. Faust in his presidential address before the American Society of Tropical Medicine in 1942, point out that some of these diseases were brought to the Western Hemisphere by the slaves which were imported, and that in the course of time some of these constituted public health problems of major importance. In this category we can point with a good deal of certainty to leprosy, yaws, yellow fever, Manson's schistosomiasis, Bancroft's filariasis, dracontiasis, and hookworm disease. Some of these, such as schistosomiasis, did not become established in the United States, but hookworm disease became and is still considered a very important public health problem in certain southern areas. For our present purpose it will suffice to review the histories of a few of these tropical diseases of the United States, citing instances of those that have become established in either limited or extensive areas, and instances of those that have not appeared in our midst for a considerable length of time.

Malaria, the most important disease in the world today, is present in the United States, and presents a major health problem in certain sections of the Southeastern part of the country and on the Pacific coast. Human malaria is caused by four species of blood parasites belonging to the genus *Plasmodium,* each producing a different type of malaria, referred to clinically as tertian or vivax malaria, quartan malaria, estivo-autumnal or falciparum malaria, and ovale malaria. The first three of these are present in the United States, vivax malaria and falciparum malaria being of most importance. Vivax malaria is more widespread and occurs in the cooler northern states as well as in the southern states, while falciparum malaria is found endemically only in the southern states.

Plague was introduced into the Western Hemisphere during the latter part of the nineteenth century, with occasional outbreaks in the United States. Recent investigations have shown that endemic or sylvatic plague exists in ground squirrels and like rodents in some of the western states, and occasionally man becomes infected from contact with these rodents.

Yellow fever first appeared in the Western Hemisphere during the seventeenth century. During the latter part of that century it appeared on the Eastern Seaboard of the United States, and during the latter part of the eighteenth century the first epidemic occurred in New Orleans. One epidemic centered around Memphis in 1878 and the last epidemic in the United States occurred in New Orleans in 1905.

Epidemic or louse-borne typhus, of rickettsial etiology, was introduced into the United States by Irish and Polish immigrants during the middle of the nineteenth century, but today no foci remain. However, Brill's disease or endemic typhus, a milder form of typhus recognized in the United States at the turn of the present century, remains with us to present an important public health problem in some of the southern and Gulf Coast states. It is present in rats and is transmitted to man by rat fleas, thus leading to the designation of "murine typhus."

Dengue fever, of virus etiology, has been known in the United States since 1824 when an outbreak occurred at Charleston, South Carolina. It is usually limited to the warmer regions of the United States, but outbreaks have occurred in Boston, Philadelphia and New York. Epidemics occurred in the southern states in 1922 and 1934, involving large population groups. Due to the widespread occurrence of the mosquito vector, *Aedes aegypti,* there is a possibility that the disease may become more widely distributed than at present.

Relapsing fever, caused by a spirochaete of the genus *Borrelia,* may be either louse-borne or tick-borne. The louse-borne type was once present in the United States, but apparently does not exist at the present time. The tick-borne type of relapsing fever, transmitted by a soft tick of the genus *Ornithodorus,* occurs endemically in several of the western states, extending from Texas and California north to Washington.

Rocky Mountain spotted fever, a rickettsial disease transmitted by ticks, formerly thought to be confined to Western Montana and limited areas in a few other states of this Rocky Mountain region, has now been reported from most of the states, including some of those on the eastern coast.

Bancroft's filariasis, caused by the nematode *Wuchereria bancrofti,* introduced into this country by the slaves, retains an endemic center at Charleston, South Carolina. The Guinea worm, *Dracunculus medinensis,* was also brought into this country by slaves, and occasionally in these days a missionary returns from Africa bringing an infection with him. This parasite has apparently not established itself in man in this country, but a worm of very similar morphology has been reported from certain wild animals, and these may possibly be reservoirs of infection.

SOME PRESENT POSSIBILITIES OF THE INTRODUCTION OF NEW OR TROPICAL DISEASES INTO THE UNITED STATES

The examples just cited will suffice to illustrate that parasitic and tropical diseases have been introduced into the United States, and that some of them have become established, creating major public health problems in some instances. Let us now survey some of the possibilities of the introduction of still other tropical diseases into the United States, or the possibilities of the already existing diseases becoming more widespread. By what mechanisms might this be accomplished? Of primary importance is the infected individual, coming into the United States and bringing his infection with him. Our fighting men are widely dispersed over the globe, many of them in tropical areas or other areas in which endemic diseases are rife. Factual data on morbidity are not available concerning the men of our present armed forces, but let us take a glance at data obtained from wars of the past, from which we may make certain inferences of importance. In the Crimean War, during the middle of the nineteenth century, the data for the year 1854 show that deaths from disease were 230 per thousand, while those occurring in action or from wounds were 150 per thousand. Cholera, dysenteries, small-pox, epidemic typhus, and fever which we now know was malaria, were listed as the major causes in the decimation of the ranks. In the World War of 1914-1918 one of the main causes of the failure of the campaign in Macedonia was malaria. Some of the battalions had 95 per cent of their officers and men stricken with malaria within a month after debarkation at Salonika. During the years 1916 to 1918 there was an average task force of 144,908 officers and men, and from this number there were 162,517 hospital admissions for malaria. During the year 1918, among a force of 128,747 there were 59,087 malaria admissions, 272 deaths and 133 cases of blackwater fever, 28 of them fatal. In 1918 the East African Expeditionary force had a strength of 58,114. Between June and December there were 50,768 admissions for malaria, with 263 deaths. Turning to the present war, there have been 13,000 hospital admissions from the British Middle East Army, and of these 45 per cent were for tropical diseases, including 735 for malaria, 18 for schistosomiasis, 5 for Kala-azar, 4,178 for dysenteries, and 1,153 for fevers of short duration.

We must face the fact that some members of the armed forces will return with tropical diseases. Malarias and dysenteries will no doubt be the most frequently encountered. We already have malaria in this country, but there will be ample opportunity for it to become more widely dispersed. New strains of these parasites, to which the population has no resistance or immunity, will be introduced. In some areas where our soldiers are fighting, the incidence of malaria is as high as 98 per cent. Falciparum malaria, usually limited to warmer climates, may become epidemic in more northern areas for limited periods, as it is introduced into a non-immune population group. This type of malaria was carried to Connecticut after the Spanish American War, and malaria appeared there in epidemic proportions for a time. Soldiers returning from Southeastern Europe at the close of World War I seeded the areas to which they returned. The mosquito vector of vivax malaria is present in the United States from the south up as far as the Canadian border, and because the vivax parasite can survive the winter in hibernating Anopheline mosquitoes, there is a possibility that this type of malaria may become endemic in a considerable number of regions of the north. On the other hand, it is not likely that falciparum malaria will become established above the Ohio river, because it does not live over winter in the mosquito.

Although amebic dysentery is present in the United States, with an average incidence of slightly over 8 per cent for the population as a whole, we can expect that soldiers will return with infections, and that the chances for the spread of this infection will increase, depending upon the factors of hygiene and sanitation. We know from recent experiences in one of our large northern cities that epidemics can occur in cool climates in areas where the level of personal hygiene and sanitation is relatively high. Bacillary dysenteries are ever present in the tropics and our troops will not remain free from them.

In passing, let us mention a few other diseases that may be brought back to this country by returning troops. Louse-borne typhus might be brought back from Eastern Europe, the Near East or North Africa. Kala-azar is present in the Near East, India, Eastern and Northern Africa, the Mediterranean region and parts of China. Oriental sore may be acquired in much the same regions. Espundia, the muco-cutaneous leishmaniasis, is limited to the New World, but many units of the Armed Forces pass through these regions or remain there for considerable time in the line of duty. African sleeping sickness, now limited to equatorial Africa, might be present among some units of returning Armed Forces. In fact, it is conceivable that persons of the armed forces and civilians that travel to tropical countries or areas in which tropical or parasitic diseases are present, might acquire any disease or diseases endemic in those regions visited. It is also possible that many of these infected persons may return to this country before they have eliminated their infection. This, in a broad sense, would constitute an introduction of the disease into this country.

The possible introduction of diseases by individuals will not terminate when this war has ended, for it is expected that the development of commercial and political relations between nations of the world will result in visits to these countries by a greater number of individuals than in any previous time in history. Large tropical areas, heretofore undeveloped, will become centers of commercial activity. Our advances in rapid transportation by boat and plane will facilitate this intercourse, and the chances that infected individuals will come or return to this country will be greatly augmented. In former days, usually enough time was spent in transit to permit the infection to become established, and cause the usual signs and symptoms which lead to its discovery. With the present rapid transportation it is possible that a person may arrive at his destination in good health, only to succumb later to an infection obtained while abroad. Dr. H. C. Clark has ably discussed the problems of disease dissemination by international transportation.

We must not forget the possibility that vectors of disease not now present in this country, may be introduced, bringing disease organisms with them to be transferred to man, or making it possible for the completion of the cycles of recently introduced disease organisms. With the advent of air traffic, this became a reality. *Anopheles gambiae,* a very efficient vector of malaria was introduced from Africa to Brazil. It multiplied rapidly, and because of its efficiency as a vector of malaria, an epidemic of considerable proportions broke out. Mosquitoes can survive airplane trips of several thousand miles, covering a span of over two days, and involving flight at high altitudes, some taking blood meals enroute. According to recent information the tse-tse fly, the transmitter of African sleeping sickness, was introduced into Brazil in this manner. At plane inspections at Natal, Brazil, during the past year, 1600 different insects were found that had been brought from Africa. *Anopheles gambiae* were found seven times. Insects that are not now important, may become important vectors of disease when introduced into another country with a different climate and where they do not face destruction by natural enemies.

Many animals are reservoirs of infections, and if brought into this country might bring an infection with them, giving it a chance to become established. It was pointed out by Cameron that pets brought into the country by returning soldiers might be instrumental in introducing a disease.

PREVENTIVE MEASURES AGAINST THE INTRODUCTION OF TROPICAL DISEASES

Despite the many possibilities for the introduction of tropical diseases into the United States, or the introduction of these diseases into new areas of the country, there are many precautions that can be taken to prevent the outbreak of epidemics and the devastating effects on health and life that they often cause. There are many avenues of approach to the varied problems that we may be called upon to face, and these need to be carefully patrolled by men well trained in the fields of preventive and tropical medicine. The responsibilities before us may be enumerated as follows: (1) protection of the Armed Forces from disease hazards, (2) solving the problems of the post-war introduction of tropical diseases, (3) assuming the responsibility for much of the public health administration of many areas of the world, both during the war and after the cessation of hostilities, (4) an evaluation of the role of tropical medicine in the development of areas in the tropics and in the development of a future place in the commerce

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of the world, and, (5) to be adequately prepared to actively participate in a program of tropical and preventive medicine.

I have assumed that the Army and Navy Medical Departments and the United States Public Health Service will play the immediate, major roles in the prevention and control of these diseases among the members of the Armed Forces. At the same time other governmental and civilian agencies are co-operating in this program of prevention of diseases among our troops. Among these may be mentioned the National Research Council, with its Subcommittee on Tropical Diseases, the Committee on Medical Research of the Office of Scientific Research and Development, the Pan-American Sanitary Bureau, the Office of Coordinator of Inter-American Affairs, the International Health Division of the Rockefeller Foundation, and many of the organizations in the fields of biology, medicine and public health. In time, the civilian physician, not now a part of any of the organized groups just mentioned, will find that he has an important part in the prevention of the spread of these diseases. National groups must collaborate with recognized international commissions. It is hoped that all of these agencies and groups will make valued contributions toward solving the problems just mentioned. We can mention with pride a number of excellent contributions that have already been made in this program against the acquisition and spread of tropical and infectious diseases. We can also mention programs in progress, and measures which we feel may be a contribution to the solution of our problems.

Early in 1941 a Division of Preventive Medicine was established in the Office of the Surgeon General of the Army, directed by Brigadier General J. S. Simmons. There are several branches of this division, including the Sanitation Branch, having to do with the control of food, water, wastes, and insect pests and vectors of disease; the Sanitary Engineering Branch which deals with the engineering aspects of sanitation; the branches of Laboratories, Military Occupational Hygiene, Venereal Diseases Control, Medical Intelligence, and Epidemiology. The Epidemiology Branch deals in the general policies relating to the control of infectious diseases. The immunization program functions under direction from this branch. All troops are vaccinated or immunized against smallpox, typhoid fever, para-typhoid fevers A and B, and tetanus. Troops serving in special areas will be vaccinated against yellow fever, typhus fever, cholera and plague. Another section of this Branch is devoted to the control of tropical diseases, under which an effective military program for the control of these diseases has been developed, including the training of specialized medical personnel to carry out the necessary control measures. "The Board for the Investigation and Control of Influenza and Other Epidemic Diseases of the Army" was established by the Secretary of War in 1941, and operates as a part of the Preventive Medicine Division. The personnel consists of 111 civilian specialists, and these are organized into ten special commissions, among which is the Commission on Tropical Diseases. The Medical Intelligence Branch collects and assembles specific information about health and disease conditions in foreign countries, which information aids in planning for measures and equipment necessary to protect our forces in foreign countries.

The Medical Department of the Navy has shared in this responsibility of the training of adequate personnel, and the development of adequate measures to meet its particular needs in the prevention and control of tropical diseases.

The United States Public Health Service, likewise has made its contribution toward the development of essential strategy against the spread of infectious diseases, and has collaborated with the Army and Navy. In December of 1942, the President created the United States of America Typhus Commission, composed of officers of the Army, Navy, Public Health Service and civilian experts, and authorized it to make a study of typhus fever and recommend measures for its control in any area affecting military personnel of the United States. The Commission is now at work in Africa and the Near East.

Early in this year an Interdepartmental Quarantine Commission was organized, composed of medical officers of the Army, Navy and Public Health Service, with the purpose of studying and revising laws designed to prevent the war-time spread of diseases into this country or between military bases abroad. The commission has already completed work in reference to the transmission of diseases and disease vectors by air transport or surface travel, and as a result of military operations. Our quarantine measures will require revision to conform to the needs that have developed with the advent of air transport, and such measures should take into consideration the international aspects of the problem of disease prevention and control and be fully supported by international agreements as to the limitations that are to be imposed. These measures should not be so strict as to impede the development of international, social, economic, political and commercial intercourse. It may also become necessary to set up a system of interstate quarantine in our own country should we be threatened with epidemics of malaria or other diseases.

We should not fail to be cognizant of the part that inadequate diet will play in the production of nutritional disease, and of the effect that this will have on the outbreak of infectious diseases as the resistance of population groups is lowered. These problems will be met by such organizations as the Office of Foreign Relief and Rehabilitation which includes officers trained in Public Health. An agency to be known as the United Nations Relief and Rehabilitation Administration is being formed, and we have other organizations such as the Inter-Allied Committee on Relief and the United Nations Organization on Food and Agriculture. These several organizations just named are interested in the physical well-being of the population of the war-torn areas, and their work should substantially aid in the prevention and control of infectious diseases among which are listed our important tropical diseases.

When the time for demobilization is reached, every effort should be made to detect the tropical diseases that will surely be brought back to this country by men of the Armed Forces. Demobilization should be conducted in such a manner that sufficient time will be available to make adequate physical and laboratory examinations which are designed to detect infected individuals as far as is practically possible. When infected individuals are discovered, they should be hospitalized, reported to their State Board of Health or placed under the surveillance of a family physician as the case merits. I have been informed by a reliable source that such plans are now being formulated by the proper authorities in our Armed Forces.

Finally, but of major importance, is the necessity for the development of adequate trained personnel to execute the many and intricate parts of these various planned or proposed programs directed against the introduction and spread of tropical and infectious diseases in the United States. To solve this problem, we must change our ideas concerning what constitutes adequate medical training. Tropical Medicine and Parasitology have never held a prominent place in the curricula of most medical schools in the United States. This inadequacy of instruction in Tropical Medicine was recognized by the leaders in this field, and has been discussed in several recent lectures and articles. The imminence of our participation in the war and finally "Pearl Harbor" awakened us to the grave need for substantial training in this subject. In October of 1941, the Association of American Medical Colleges appointed a committee to make a study of the teaching of tropical medicine in medical schools. A report was prepared which showed that only a small minority of the schools were giving adequate instruction in either parasitology or tropical medicine, and that many of them lacked instructors qualified to teach these subjects. With renewed interest in these subjects the Markel Foundation made an appropriation to the Association of American Medical Colleges to enable instructors from the United States and Canadian medical schools to attend the intensive courses in tropical medicine at the Army Medical School and Tulane University. Each medical school was offered the opportunity of sending a clinical instructor to the Army Medical School and a pre-clinical instructor to Tulane. Both courses were fully enrolled at the initial sessions and a number of applicants had to be deferred until later sessions.

In 1941, at the suggestion of the Subcommittee on Tropical Diseases of the National Research Council, the Army Medical School established this special two-months course for medical officers, with a faculty composed of authorities both within and outside of the government services. After the Secretary of War issued an order that every unit of troops going to the tropics should be supplied with a medical officer trained in tropical medicine, the course was expanded to accomodate two hundred students from the Army Medical Corps plus a number of instructors from medical schools. The Department of Tropical Medicine at Tulane University has also given similar instruction every two months to an additional thirty medical officers. Other branches of the armed forces have given similar training in tropical medicine, or closely allied subjects, to medical officers, and various governmental departments have provided special training for members of their personnel, designed to prepare them for work in the control of tropical diseases.

Some medical schools added an instructor of parasitology to their teaching staffs, and the instructors who had been enrolled in the special courses in tropical medicine returned to their schools to initiate new courses in parasitology or tropical medicine, or to expand the existing courses by the addition of pertinent subject material. In these courses they are teaching students, the majority of whom will enter the Armed Forces following graduation and the completion of an internship, and it is hoped that the majority of these will return to practice medicine in this country, or will offer their services in other countries where the need for physicians is imminent. It is a fortunate coincidence that these medical men, many who will have had experience in the tropics, will be released from the Medical Corps concurrently with men from other branches of the service. It is expected that their services will be in great demand, because, regardless of the number of infected individuals that are known to return to this country, we may expect to be faced with cases of disease that appear after delayed incubation periods, or instances of relapses that may occur months or years following the initial period of symptoms.

It would seem desirable that physicians now in practice obtain additional information and experience in tropical medicine, whenever opportunity affords. It has been pointed out repeatedly by persons of authority, that medical men will not be able to cope with some of the diagnostic problems with which they are confronted, unless they have a basic knowledge of tropical medicine and are aware of the possibility that some of these diseases may have had an origin in tropical countries. As an aid to correct diagnosis, there will be necessity for an additional number of clinical laboratories to establish the specialized services demanded for a laboratory diagnosis of tropical diseases, staffed with technicians that have had sufficient specialized training and practice to make them competent in carrying out the necessary diagnostic techniques.

In conclusion, let me reiterate that it will most likely *not* be possible to prevent the introduction of tropical or new diseases into this country, i.e., the introduction of infected individuals, but if we diligently and wholeheartedly apply our present knowledge of tropical and preventive medicine both from the standpoint of the individual and that of the universal public, these cases should not lead either to the establishment of extensive endemic foci or to the outbreak of severe epidemics. As these problems confront us, it is hoped that we will deal with them rationally, and without the frantic anxiety that has been displayed by some popular writers who have expressed their opinions on the subject. It is hoped that the lessons which we learn from international collaboration in the prevention and control of diseases, tropical or others, may be transferred to the cause of building a permanent peace. Ironically enough, the evils of tropical diseases may be instrumental in the development of a period of peaceful, healthful living not previously experienced by mankind.

The Management of Renal Calculi

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In the last ten years progress in the management of patients with renal lithiasis has been pronounced. This has not been accomplished primarily by improved surgical technics but rather by more complete investigation of the etiologic factors associated with stone formation, their correction during the operative procedure, or their eradication during the period of postoperative observation.

Recurrence of renal calculi after conservative operations upon the kidney for renal lithiasis has constituted one of the most serious complications in this field of surgery.

In the literature the incidence of recurrent calculi varies considerably, the incidence ranging from 10.79 to 45 per cent. With such a stated morbidity it is immediately evident that the operative procedure per se must be considered as only one phase in the management of individuals with renal lithiasis. Unless additional therapeutic procedures are employed, recurrent calculus formation may be anticipated, for as a general rule the causative factors instrumental in the production of the primary calculus are not eliminated by surgical removal of the kidney stone. At the present time several etiologic factors seem to be definitely associated with the formation of renal calculi.

ETIOLOGY

Hyperparathyroidism. In 1934 Barney and Mintz reported a series of 18 cases in which a diagnosis of hyperparathyroidism had been established and substantiated by surgery. In 11 cases (61.1 per cent) calculi were present in the urinary tract. In the cases studied at the Cleveland Clinic hyperparathyroidism has been associated with renal calculi in less than 0.1 per cent.

In view of the previously mentioned reports, however, it is essential to determine the serum calcium and phosphorus level of the blood in all cases of renal lithiasis. If blood calcium level is elevated and blood phosphorus level is lowered, further roentgenographic studies of the bones and investigations of the calcium balance are advisable.

Deficiency in the Diet. Numerous clinical and experimental observations have demonstrated the relationship between deficiencies in the diet, or a poorly balanced diet, and stone formation.

Stone areas (regions in which calculi are unusually prevalent) have been reported in several countries. The inhabitants in these localities have been found to subsist on poorly balanced diets. Following World War I calculus disease definitely increased in certain parts of Europe. Geza Illyes stated the increase occurred in countries where the inhabitants subsisted on poorly balanced diets for extended periods of time. Joly attributed the decline of calculus disease in childhood to the dietary and nutritional progress in England and France. Biophotometric studies revealed that 68 per cent of patients with renal calculi seen at the Cleveland Clinic had a positive test for vitamin A deficiency. Pyrah and Fowweather reported an incidence of 40 per cent, while Ezickson and Feldman found pathologic adaptation from a mild to severe degree in 24 of 25 patients with urolithiasis.

In laboratory animals I have been able to produce calculi composed of the same constituents as those observed in clinical patients. The animals were maintained on various diets deficient in varying degrees of vitamin A for extended periods of time.

Vitamin A should be prescribed postoperatively not only to correct vitamin A deficiency if present but also for its specific effect on epithelial structures.

Infection in the Urinary Tract. The relationship between infection and the formation of renal calculi has long been recognized. However, that renal infection is not the only causative factor is evidenced by the fact that many patients with small renal calculi do not have pus in the urine, and a sterile culture of the urine is secured from the kidney by ureteral catheterization. It is important, however, that a gram stain be made of the urinary sediment in each case.

In addition to routine cultures, it should be determined whether or not the organism possesses the power of splitting urea with the resultant formation of ammonia and carbon dioxide. If a urea splitting organism is present, the use of ammonium salts as acidifying agents is contraindicated. Approximately 18 per cent of the Staphylococci and 11 per cent of the Bacillus coli, as well as the Proteus and Micrococcus urae, have been found to possess this power. It is thus evident that complete bacteriologic study should be made in all cases, not only to treat the infection scientifically but also to prevent the recurrent formation of renal calculi.

Rosenow and Meisser in 1921 demonstrated the specificity of Streptococcus in the formation of renal calculi. In view of their observations foci of infection in the teeth, tonsils, prostate, and cervix should be eradicated in all patients with renal calculi.

Stasis. In certain patients with renal lithiasis urostasis definitely seems to be a contributing factor. Stasis is conducive to the shifting of the Ph of the urine to the alkaline side and fosters the introduction of urinary tract infection. An intravenous urogram before operation provides information as to the nature of the obstruction, which may be removed at operation.

Metabolic disturbances such as gout, cystinuria, oxaluria, or permanent noninfected phosphaturia require study in the preoperative period in order that corrective dietary measures may be instituted postoperatively. To briefly reiterate, therefore, preoperative study should include (1) study of possible foci of infection including the prostate, teeth, tonsils and cervix; (2) classification of the organism present in the urinary tract, i.e., a gram stain of the urinary sediment, cultures, and a study of urea-splitting properties of the organism; (3) blood chemistry studies for hyperparathyroidism with studies of the calcium balance, and roentgen studies of the bones if indicated; (4) biophotometric test for vitamin A deficiency; (5) an intravenous urogram to determine the presence of stasis; (6) metabolic studies including determination of the blood uric acid; (7) determination of the type of phosphaturia; (8) determination of the type of crystals occurring in the urine. Minimizing the significance of such investigations will result in a high increase of recurrent calculus formation.

TREATMENT

In recent years a more conservative attitude has been taken toward the surgical management of patients with renal lithiasis. Refinements in surgical technic and the introduction of newer chemotherapeutic agents to eradicate kidney infections have permitted such conservatism. In the past nephrectomy was occasionally recommended in individuals in whom today an extensive nephrotomy would be advised. Various procedures are available for the removal of a stone from the kidney after the organ has been adequately exposed.

Calculi confined to the kidney pelvis usually may be removed by pyelolithotomy. This is the simplest procedure and is accompanied by a minimal amount of trauma. After removal of the stone the incision in the kidney pelvis is closed with 000 plain catgut to minimize urinary drainage on the loin, which is conducive to the introduction of a Staphylococcus albus infection into the kidney. Nephrolithotomy should always be as conservative as possible. I believe the incision into the renal parenchyma should be made with a cutting current rather than with a scalpel. In the laboratory we have demonstrated that the immediate bleeding is less profuse, and less infarction and atrophy of renal parenchyma are produced with a cutting current. In nephrotomy a number 18 F dePezzer catheter is inserted routinely through the cortex down to the kidney pelvis for drainage, irrigation, and removal of retained clots.

Under certain circumstances a heminephrectomy is a conservative procedure. In the presence of a pyocalyx containing a calculus associated with a stricture of the infundibulum, resection of the upper or lower pole of the kidney is the procedure of choice. In general, the procedure accompanied by a minimum of trauma and injury to renal tissue is advisable.

Postoperatively, close observation is essential for an extended period of time. The primary aim is to avoid the recurrent formation of stones. After eliminating the causes of stone formation indicated by preoperative study, a diet is prescribed depending upon the chemical constituents of the stone removed. A high vitamin A, acid or alkaline ash diet with modifications is utilized to control the pH of the urine and to maintain the urinary salts in complete solution. By maintaining the urinary salts in complete solution, precipitation and formation of crystals are avoided, thereby eliminating a requisite necessary for stone formation.

Patients are taught to determine the pH of the urine and to report to the physician at frequent intervals. Thus if the pH of the urine is not being maintained at the proper level, further changes in the diet may readily be made. By using diet in conjunction with other established procedures, we have reduced the incidence of recurrent calculus formation from 16.4 to 4.9 per cent. That dietary measures are effective not in our hands alone is evidenced by reports in the literature citing progressive decrease in the incidence of recurrent calculus formation.

In view of results being secured at present, we feel justified in placing certain patients with renal calculi on a diet to dissolve the calculus without recourse to surgery. This type of treatment, however, is not applicable to all cases. Cystine stones seem to respond to dietary dissolution more rapidly than any other type, while oxalate calculi are the most resistant to dietary treatment. Similarly, phosphatic and carbonate stones respond more readily than those composed of uric acid. If the calculus is in the ureteropelvic junction and is producing obstruction and thereby damaging the renal parenchyma, or if the pH of the urine cannot be controlled, dietary treatment should not be instituted. The patient cannot be treated in a haphazard manner. The diet is explained to them in detail by a dietitian. The diet must be followed by the patient as carefully and conscientiously as a diabetic patient follows the diabetic diet. The dietary regimen is used routinely in patients who have previously passed stones spontaneously and in whom recurrent stone formation is to be avoided.

SUMMARY

Removal of a calculus from the kidney is only one part of the management of renal lithiasis. Eradiacation of the causative factors instrumental in the production of the primary stone and control of the pH of the urine by dietary means are essential in the prevention of secondary stone formation.

The dietary regimen may be used to secure dissolution of a renal calculus in a selected group of patients, and to prevent further stone formation in individuals who have passed stones previously.

The Talcum Problem in Surgery and Its Solution

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INTRODUCTION

The purpose of this report is to re-emphasize the very serious surgical hazard incident to the use of talc as a dusting powder for rubber gloves, and to recommend potassium bitartrate as a substitute powder.

Even a cursory knowledge of the rapidly increasing surgical literature dealing with talc convinces one that this powder creates serious postoperative hazards. After the technique of dry gloves was adopted, practically a quarter of century elapsed before surgeons recognized the evil agency of talc, and even now many are unaware of its harmful potentialities. Equally noteworthy is the fact that in the various reports devoted to this subject, efforts center almost exclusively on detailed descriptions of the surgical complications due to talc, with practically no stress laid on the possibility of substituting for it some satisfactory, innocuous powder. As a result, at this very moment talcum powder is in almost universal use in preparing gloves aseptically.

There are numerous reports in the literature, in which are recorded cases of postoperative intestinal obstructions due to talc. In our survey of the literature we found numerous case histories of patients who were subjected to from two to four and even more operations for the relief of successive intestinal obstructions due to talc peritonitis. Even the most careful rinsing of gloves in water or alcohol, before operating, does not free them from talc. Then too it is not at all uncommon for as much as 100 milligrams of talc to spill out of an accidental tear in a finger tip.

Studies by Weed and Groves show that: (a) in 74.4 per cent of all operations some gloves are torn, and (b) that 22.6 per cent of all gloves used show rents or punctures. These data were obtained by Weed and Groves through a period of 20 months of operating, using a total number of 35,763 gloves. Some months 32 per cent of the gloves showed rents or punctures. The high incidence of perforation of rubber gloves during operation (22 per cent) was recently emphasized in an Editorial in The British Journal of Surgery. German found that in a series of 50 patients, 40 showed intraperitoneal talc that had been deposited accidentally during a previous laparotomy. German examined his microscopic preparations in polarized life, which renders the talc crystals much more readily visible. Forty-two of German's patients had had from 1 to 7 previous laparotomies. Of course in such a study the talc may come from the surface of the gloves or through accidental perforations of them, or possibly from gauze sponges or pads contaminated with talcum powder.

The noxious properties of talc are thrown into bolder relief when one learns that it is a trouble maker elsewhere than in the peritoneal cavity. Granulomata resulting from talc implantation have been described in the rectum, vagina, cervix, brain and in healing wounds, by Antopol and Robbins, Erb, Antopol, Fienberg, Ramsey and Douglass, Grieco, Cline and others. We have heard, by way of personal report, of troublesome consequences following the accidental entrance of talcum powder into the conjunctival sacs of operating room nursing personnel. On the basis of a study of the talcum hazard in the preparation of rubber gloves for sterilization, made by the Illinois Division of Industrial Hygiene, Kronenberger suggests that it would be wise to institute measures to control and remove talc dust in order to avoid health risk to nurses.

HISTORICAL RESUME

The earliest reported clinical work on intra-abdominal complications arising from powder in the peritoneal cavity had to do *not* with talc, but with lycopodium, which is a dusting powder made from the club mosses, to the spores of which the peritoneum reacts almost exactly as it does to talc. Hippolyte Martin in 1881 as well as Muscatello were the first to demonstrate experimentally the ill effects of lycopodium powder on the peritoneum. In 1913 Lambert showed that even in cell growth *in vitro,* lycopodium spores evoked a foreign body response with outgrowth of connective tissue cells, wandering cells and giant cells. Marchand of Leipzig in 1921 corroborated experimentally the severe irritant qualities of lycopodium powder when injected intraperitoneally in animals. During the past ten years considerable experimental work has been reported emphasizing the irritant qualities of talc when injected intraperitoneally, intrapleurally, intrapericardially or subcutaneously. In fact in a certain type of cardiac disease talc has been recommended to produce pericardial adhesions in an attempt to promote better collateral circulation.

In 1923 Roth published the first contributions to the clinical chapter of postoperative complications arising from dusting powders. In this instance the dusting powder was lycopodium, spilled from a glove which tore during a laparotomy. The results of this accident were three subsequent laparotomies for adhesive intestinal obstructions, supposedly due to tuberculosis (many miliary nodules were found on the peritoneum) until pathological examination disclosed that the milia represented foreign body reactions to spores of lycopodium. In 1933 Antopol reported six cases of complications resulting from lycopodium spores in the urinary bladder, testicle, kidney, peritoneal cavity, neck and female breast. In the same paper Antopol recorded the first case of intraperitoneal granuloma due to talc. Antopol's description of the histopathology of the lesions resulting from the contamination of the peritoneum with these dusting powders remains as accurate today as when it was written, ten years ago. In the instance of lycopodium the offending agents are the lycopodium spores, whereas in the instance of talc, they are the talc crystals (predominantly magnesium silicate). The spore or the talc crystal forms the center of a tubercle-like structure, made up of lymphocytes, epitheloid cells and giant cells (of both foreign body and tumor giant cell types). In our experimental animals we found all types of adhesions, ranging from a massive occlusion of almost the entire peritoneal cavity, to less massive localized adhesions of stomach, spleen, liver and omentum or intestinal loops, or merely thin adhesive bands of fixed omental strands that not infrequently caused death from intestinal obstruction in our animals.

Any attempt to find a substitute for talc must be based upon two fundamental considerations: (1) The substitute powder must possess such a degree of actual or potential *solubility* as to be disposed of rapidly and completely by some form of peritoneal or tissue absorption. If this be not true, the powder collects in the peritoneal cavity as masses or plaques which induce foreign body granulomata, which in turn result in adhesions; or the powder sets up a foreign body reaction in wounds or on mucous membranes; (2) The powder must possess such a degree of *insolubility* as to withstand steam sterilization without losing that dusting property which serves to prevent the glove surfaces from adhering.

One of us (D. J. V.), with the foregoing considerations in mind, but nevertheless, on solely empirical grounds, selected potassium bitartrate as fulfilling the necessary requirements. This powder is sufficiently insoluble (0.37 grams per 100 cc. of water at 0° C. and 6.1 grams per 100 cc. at 100° C.) to preserve its powdering-effect after autoclaving. Studies of the pharmacologic action of the tartrates in man and experimental animals, by Underhill and his co-workers, Salant and his co-workers, Post and others, reveal that only large doses are toxic. It has been shown that the toxic dose in the experimental animal is 0.5 gram per kilogram of body weight administered subcutaneously, or about one and one-fourth ounce (35 grams) in a man weighing 150 pounds. Our studies show that in a carelessly powdered glove as much as 100 mgms. of tartrate may collect in each fingertip. Assume, for the sake of argument that gloves have been carelessly prepared and that during an operation ten finger ends were torn, spilling 1000 mgms. of potassium bitartrate in the peritoneal cavity, there then would have been scattered only 1/35 of the toxic dose for animals. One does not have to worry about the bitartrate powder on the surface of the gloves, because, unlike talc, it washes off easily and completely, before operation.

MATERIALS, METHODS AND RESULTS

We studied twenty-four different powders as follows: calcium carbonate (precipitated), calcium gluconate, calcium citrate, calcium phosphate (dibasic), calcium phosphate (tribasic), calcium tartrate, calcium d-malate, magnesium bicarbonate, magnesium carbonate, magnesium phosphate (dibasic), magnesium-ammonium-phosphate, magnesium tartrate, potassium bicarbonate, sodium bicarbonate, sulfanilamide, talcum and potassium bitartrate. Seven different forms of starches were also tried, as follows: cornstarch, arrow-root-starch, soluble starch, rice starch, Linit starch*, special starch*, and amylocellulose**. These studies were directed toward determining primarily, whether or not the various powders possessed such dusting properties as to permit the gloves to come out of the process of steam (autoclave) sterilization in adequate condition. Many of the powders we tried met this test successfully because most of them are sufficiently insoluble. With the starches however, we were baffled by the gelatinization which resulted from sterilization and rendered the gloves unusable.

The second step consisted in testing the physically satisfactory powders on white rats (150 gms. to 350 gms. in weight) by injecting sterile watery suspensions of varying concentrations (usually 2 cc. of 5 per cent solution) into the peritoneal cavities. We made in all 194 experiments on 194 individual rats. At intervals varying from 5 minutes to 24 weeks after injection the rats were sacrificed, autopsies performed and gross and microscopic studies made of pathological lesions.

The results of the experiments may be summarized very briefly by saying that *with one exception,* every powder that successfully met the test of steam sterilization produced pathological lesions of one type or another in the peritoneal cavities of the test animals, i.e., powder plaques, granulomata, adhesions or peritoneal serous exudates. The one exception was *potassium bitartrate* which whether it was strewed on the abdominal viscera through a laparotomy wound or injected into the peritoneal cavity as a watery suspension in varying strength, was rapidly disposed of without causing even the slightest untoward toxic or physical effects—local or general—in any of our experimental animals.

It is a notable fact that bitartrate of potassium of a mesh of from 210 to 270 seems to powder gloves better than does the finer mesh powder. We therefore recommend the coarser mesh (210 to 270) which in reality, is the commercial product (cream of tartar) even though it feels a trifling bit more gritty to the hands of the surgeon.

As a practical test, we have placed the bitartrate powder in the operating rooms of the Washington University School of Medicine (Barnes Hospital), The Jewish Hospital, De Paul Hospital and our own Barnard Free Skin and Cancer Hospital. When the standard technique for rubber glove sterilization was followed (subjecting the powder, either in gauze sachets or in metal or glass shakers and subjecting likewise the powdered gloves to 15 pounds of steam pressure for 15 minutes) we got unanimously favorable reports. We find that when properly sterilized we can repeat the sterilization of the same batch of bitartrate more than half a dozen times without causing any deterioration of the powder. On the other hand either too much heat or too long an exposure to the proper degree of heat, ruins both the gloves and the dusting effect of the powder, discoloring and caking it. In this connection it is important to remember that in contrast to talc with its heavy bacterial and spore content, the bitartrate is actually bacteriostatic and relatively innocuous, from the point of infectivity, even before sterilization.

In attempting to determine the effect of potassium bitartrate on pyogenic bacteria, agar plates were seeded by pouring an evenly dispersed broth suspension of staphylococci (Staphylococcus aureus) and B. Coli over the plate surface.

We found that the bitartrate had a marked bacteriostatic effect on the growth of both staphylococcus aureus and the colon bacillus on agar and in broth cultures. Talcum, of course, not only shows no antibacterial action but, on the contrary is heavily laden with pathogenic bacteria and resistent spores.

CONCLUSIONS

1. Talcum powder is, under any circumstance, a grave menace in surgery. Once having gained entrance into the animal organism this powder sets up a reactionary, productive inflammation that is permanent, progressive and that may be provocative of almost insuperable complications. Furthermore, postoperative residual talcum has been demonstrated by one investigator in various intra-abdominal viscera in 80 per cent of patients he examined.

2. It is certain that even meticulous care in washing off the surface of rubber gloves before operating does not guarantee against contamination of the operative field with talc.

3. The difficulty in substituting for talc lies in the fact that an insoluble powder like talc (hydrous magnesium silicate) sets up a granulomatous foreign body reaction, where as a soluable dusting powder dissolves during the process of sterilization of the gloves, rendering them adherent and thus difficult or impossible to put on. Dry sterilization of the gloves might be regarded as a solution of this difficulty, but it does not meet the rigid requirements of aseptic

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*Courtesy of The Corn Products Refining Company.

**Courtesy of The Corn Starch Division of Anheuser-Busch, Inc.

surgery and is highly destructive to the life of the gloves. Boiled gloves are sloppy and generally undesirable.

4. Potassium bitartrate meets the physical requirements imposed by steam sterilization. It is readily and harmlessly disposed of by the body tissues and fluids. It causes no consequent peritoneal adhesions. Since, therefore, there are no demonstrable risks attendant upon its use, it may be recommended as a substitute for talc in powdering rubber gloves. We have received no complaints regarding undesirable skin effects.

5. Potassium bitartrate possesses a certain degree of bacteriostasis for the colon bacillus and the staphylococcus aureus.

6. It is important that in the process of sterilization the bitartrate of potassium should be subjected only to the now standard and accepted technique for sterilizing rubber gloves, namely, 15 minutes of autoclaving under 15 pounds of steam pressure.

Endometriosis and Its Serious Complications

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In any study of endometriosis adenomyoma and diffuse adenomyosis of the uterus must be included as the pathologic picture is the same in each condition, namely, endometrium-like tissue in an ectopic location.

ETIOLOGY

The etiology of endometriosis is still a controversial question and various theories have been advanced. In 1860, Rokitansky published an article on adenomyoma of the uterus and in 1896, von Recklinghausen advanced the theory that adenomyomas probably developed from embryonic rests of the mullerian ducts or the mesonephros. About the same time Cullen suggested that adenomyomas of the uterus and diffuse adenomyosis of the uterus developed as the result of invasion of the uterine wall by endometrial cells that continued to grow in their new location at the expense of the normal myometrium. This theory is generally accepted as the explanation for the origin for adenomyomas in the uterine wall. In 1897, Pfannensteil reported the first case of adenomyoma in the rectovaginal septum. In 1899, Russell reported the first case of endometrial tissue in an ovary and he attributed it to a fetal rest of mullerian ducts.

The serosal theory or theory of metaplasia advanced by Iwanoff has many supporters among leading gynecologists and pathologists, such as Robert Meyer and Novak, in this country and most continental European authors. According to this theory any tissue derived from the celomic mesothelium can change its structure as the result of an inflammatory process or because of some hormonal stimulus. In the case of the germinal epithelium of the ovary, it may change from cuboidal cells to cylindrical cells that closely resemble normal endometrial cells. Meigs has suggested delayed marriage and infrequent childbearing with excessive and prolonged stimulation of the celomic epithelium by estrin and progestin as predisposing causes which probably account for the increase in the number of cases encountered at present especially among private patients whose marriages are, as a rule, later than those of the ward patients.

The implantation theory of Sampson published in 1921 is based on his assumption that viable endometrial tissue, as a result of retrograde menstrual flow through the fallopian tubes, becomes grafted on and continues to grow and spread on the surface of the ovaries or any part of the pelvic peritoneum. It is possible that in some cases endometrial tissue does pass through the tubes before they become occluded but in most of the cases in which we performed operations we have found blood in the cul-de-sac. It has not been typical menstrual blood but rather has been the result of hemorrhage from the wall of a ruptured ovarian cyst. Both Sampson and Halban have advanced the theory that in some cases endometrial cells are carried by the lymph or blood stream and by this method true metastasis of endometrial tissue is possible to distant parts of the body, such as the labia, lungs, arms, thighs, and so forth. Implantation of endometrial tissue into the abdominal wall following operation in which the uterine cavity is opened or the tubes are divided is referred to in all treatises on the subject and must be accepted as the etiologic factor of endometriomas in abdominal wounds.

It is probable as Ewing has suggested that similar tumors included at present under the same name by various authors have entirely different origins. Most of the literature on this interesting subject has had to do with the etiology or pathology but the diagnosis of the condition, treatment and results following treatment are of most importance to the patient and her surgeon.

DIAGNOSIS

At present, a preoperative diagnosis of adenomyoma, ectopic adenomyoma, endometrioma or endometriosis is made frequently. The symptoms, however, vary so much that a correct preoperative diagnosis is not possible in a high percentage of cases.

The factors that, if present, will help most in making a correct diagnosis are as follows: (1) the age of the patient; endometriosis is most common between the ages of twenty-five and forty-five years; (2) dysmenorrhea of the acquired type; (3) pelvic discomfort, often extending to the sacrum, into the rectum, or down the thighs, which is always worse at the time of menstruation; (4) menorrhagia; (5) sterility which may be absolute but more often is relative and (6) dyspareunia. If the pelvic portion of the colon is involved, pain, constipation and possibly rectal bleeding at the time of menstruation may occur.

The findings on bimanual pelvic examination vary with any associated pathologic condition, and with the extent and situation of the disease. A frequent finding is adhesions which cause fixed retroversion of the uterus, with or without recognizable tumor; other common findings are thickening in the adnexal regions and tenderness on pressure. Fibromyomas are associated with endometriosis in about 80 per cent of cases. In some cases cysts several centimeters in diameter develop in the ovaries. Small, irregular nodules or thickenings of the rectovaginal septum, or in the cul-de-sac, best felt on rectal examination, are highly suggestive of endometriosis.

Proctoscopic examination and roentgenologic examination after administration of barium are of comparatively little help in diagnosis of endometriosis of the bowel and as a rule only confirm the diagnosis made on a combined abdominopelvic examination of an extrarectal mass.

Incidence, Site and Various Other Features

The incidence of endometriosis is hard to determine, for many of the tumors are small and are not recognized by surgeons or pathologists in routine examination of removed tissue. Green-Armytage reported an incidence of endometriosis of 8.9 per cent in a series of 1,000 consecutive abdominal operations. Sampson found 21.6 per cent in 296 laparotomies and was of the opinion that from 10 to 20 per cent of all women between thirty years of age and the menopause have endometriosis.

Heterotopic endometrium or tissue resembling endometrium has been reported (Fig. 1): (1) in the uterus, where the growths may be diffuse or discrete; and (2) outside the uterus. Lesions outside the uterus frequently are in the ovaries, rectovaginal septum, uterine ligaments, fallopian tubes, abdominal wall, umbilicus, groin, intestinal wall, wall of the bladder, or the vagina. Cases of endometriosis of the lungs, labia, leg and arm have been reported also.

There is no doubt that as a gynecologist becomes interested in this subject he recognizes endometriosis more and more frequently on exploring the pelvis. Many of the tumors found on the pelvic peritoneum of middle-aged women, however, are small and we are satisfied they seldom cause symptoms. On the other hand, many patients come to operation as the direct result of ectopic endometrial growths, many of which interfere with the normal life of the patient and some of which are a real danger to life. The younger the patient when symptoms of endometriosis develop, the greater the chance that serious complications will develop. In fact, the treatment of the young woman who has endometriosis is always a cause for worry. If any ovarian tissue is left, she is almost certain to continue to have an increasing amount of trouble and if castration is performed, she will have more or less trouble as the result of premature menopause. Fortunately, under modern medical management much can be done for these patients, with comparatively little risk of extensive growth of the ectopic endometrial tissue. The rate of growth of endometriomas varies considerably in different patients and the age at which they start also varies. When the bowel becomes involved, the degree of obstruction varies with the duration of the lesion and its location. In general it can be stated that if the patient reaches menopause before definite obstruction develops she will not have any further trouble. Locally, endometriomas are invasive in the same way that carcinoma is but they cease all invasive qualities and shrink up or entirely disappear when ovarian function stops and metastasis is a rare sequela. We must ever keep in mind, however, the possibility of cancer developing in any of these tumors containing endometrial-like cells. I believe it is more than probable that some of the adeno-carcinomas of the ovaries and other pelvic organs have their origin in ectopic endometrial tissue.

Mayo Clinic Series

From January 1, 1923 to September 30, 1943, a diagnosis of endometriosis was made at the Mayo Clinic in a series of 2,062 cases. More than 500 of these cases were of unusual interest or presented serious complications. More than 84 per cent of the patients were between thirty and forty-nine years of age. More or less complete destruction of the ovaries by tarry cysts several centimeters in diameter was the most frequent indication for radical operation. In this series of cases the rectovaginal septum, rectum, rectosigmoid or sigmoid was involved in 329 cases. Other sites of involvement are given in Table 1.

TABLE 1.

THE LOCATION OF ENDOMETRIOSIS IN 2,062 CASES
January 1, 1923, to September 30, 1943

Organ or part involved (or adherent)	Number
Uterus	1,415
Ovary	642
Pelvic peritoneum	276
Sigmoid, rectum and rectosigmoid	266
Fallopian tube	142
Ligaments of uterus	133
Cul-de-sac	110
Bladder or peritoneum of bladder	52
Rectovaginal septum	63
Vaginal wall	39
Cervix	32
Abdominal wall	32
Small intestine	26
Cecum	14
Umbilicus	9
Appendix	7
Ureter	6
Femoral hernia	2
Labia	1
Vesicovaginal septum	1
Total organ or part involved	3,268
Total cases	2,062

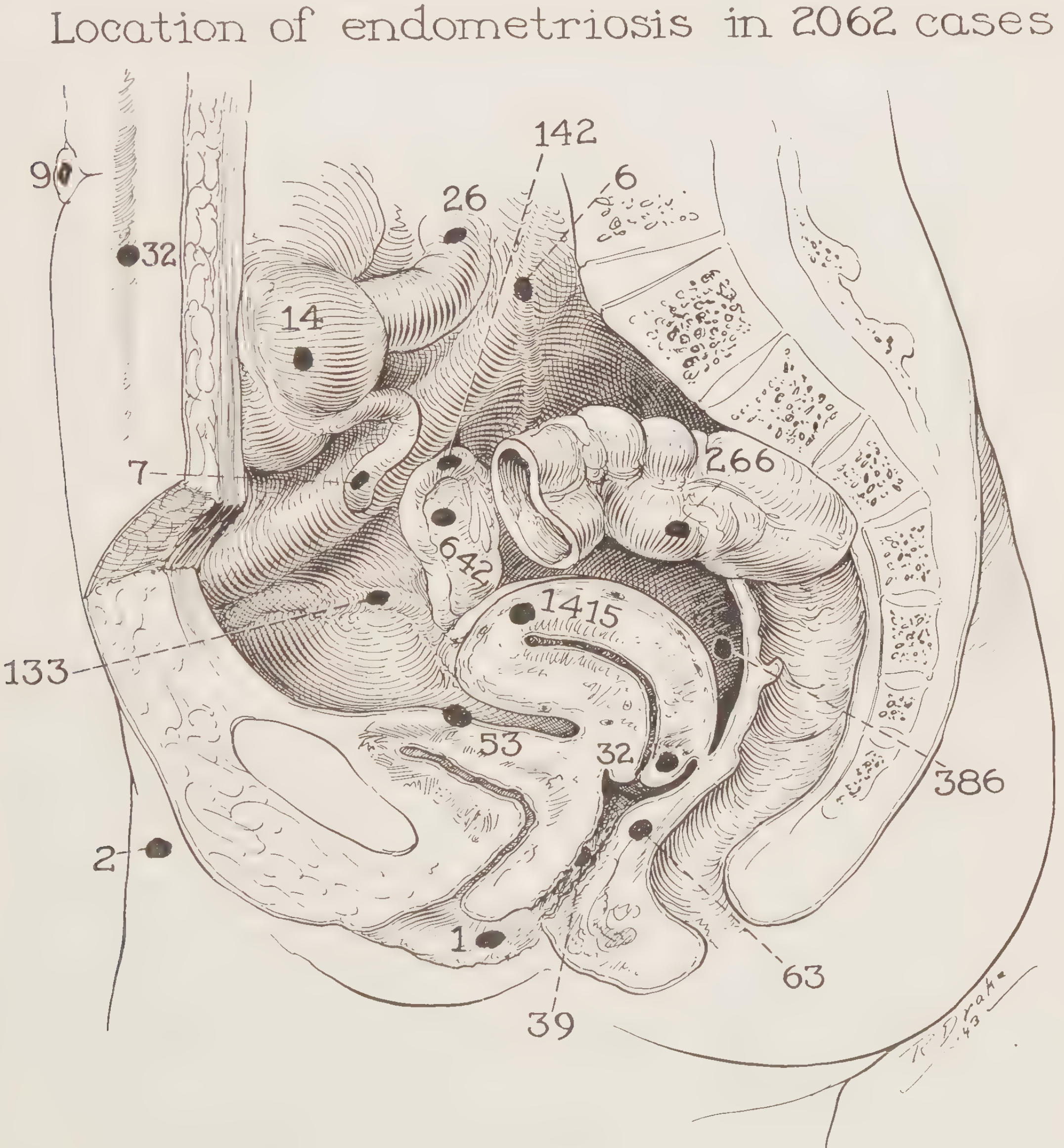
Location of endometriosis in 2062 cases
142
9
26
6
32
14
7
266
642
1415
133
53
32
386
2
1
63
39
T. Drake '43

FIGURE 1.

Report of Six Cases with Complications

Case 1. The patient, a married woman, aged thirty-two years, had never been pregnant. In 1940, two years previous to admission to the clinic, the right tube and ovary had been removed because of pain in the left lower quadrant of the abdomen. At operation a tarry cyst of the ovary was found. In November, 1941 she was admitted to a hospital elsewhere because she had not had a bowel movement for two weeks. She was not, however, acutely ill. On proctoscopic examination and roentgenologic examination of the colon an obstruction of the sigmoid at a level of the pelvic brim was diagnosed. After a saline laxative she became acutely ill and symptoms of obstruction appeared. The abdomen was opened and what was thought to be a malignant lesion was found to be obstructing the lower part of the sigmoid. Colostomy was done. Her general health became fairly good, but the colonic stoma did not function well.

On pelvic examination at the clinic in April, 1942, the cul-de-sac and uterosacral ligaments were found to be nodular and indurated. There was a napkin-ring type of lesion in the sigmoid. Proctoscopic examination revealed a mass outside the anterior wall of the bowel which impinged on the lumen. The overlying mucosa was adherent. The caliber of the lumen was decreased and mobility was diminished.

On opening the abdomen on April 28, 1942, an adenomyoma of the sigmoid which was causing obstruction and was firmly adherent to the uterine cervix was found. Because of the possibility of a malignant lesion the left tube and ovary and 6 to 8 inches (15 to 20 cm.) of the sigmoid were removed en masse. Total hysterectomy and appendicostomy were performed. The latter was done because the previous colonic stoma was not functioning well. The sigmoid was anastomosed end to end. The pathologist reported chronic metritis, polypoid endometrium of the uterus, chronic cervicitis, chronic salpingitis, perisalpingitis, chronic cystic oophoritis and peri-oophoritis. Many inflammatory lesions and endometrial growths were present on the posterior surface of the uterus. In the sigmoid was a localized region of adenomyosis which partially encircled and narrowed the lumen of the bowel. The sigmoid was densely adherent to posterior wall of uterus.

On the sixth postoperative day flatus began to pass through the rectum. On the tenth day fecal drainage appeared through the vagina, but when she left the hospital on the thirtieth day the rectovaginal fistula and the appendiceal stoma were closed and fecal drainage was occurring through the rectum and the old colonic stoma. Since dismissal her health has become good and she plans to return for closure of the colonic stoma this fall.

Case 2. The patient, a married woman aged thirty-seven years, had never been pregnant. The past history included appendectomy and right oophorectomy for an inflamed ovary in 1930, myomectomy in 1936, and an operation on the left ovary for cyst in March 1943. She had pain in the left lower quadrant of the abdomen prior to the operation in March, 1943. On the seventh day after that operation fecal material appeared in the incision and colostomy was performed for intestinal obstruction.

The patient was admitted to the clinic on August 16, 1943, with the complaint of a draining abdominal sinus which had been present for five months and a poorly functioning colonic stoma.

On examination a midline scar with a colonic opening 1 to 2 cm. in diameter was found. A mass in the pelvis was adherent to the bowel wall. Roentgenologic examination of the colon revealed an external fistula which involved the middle portion of the sigmoid. Considerable constriction and fixation of the bowel were noted at this point. Proctoscopic examination revealed that the bowel was adherent anteriorly to an extrarectal mass.

Operation was performed on August 24, 1943. It was found that practically all the contents of the bowel emptied through the abdominosigmoidal fistula. The old scar and fistulous tract were excised. The sigmoid was found to be practically divided. There were many firm adhesions among a tarry cyst of the left ovary, several loops of small bowel and another loop of sigmoid. The adhesions were separated and the remaining portion of the left ovary removed. After the sigmoid was freed, it was thought best to remove 3 inches (7.5 cm.) of it and perform end to end anastomosis. The cul-de-sac was blocked by stitching the fundus of the uterus to the posterior brim of the pelvis. Five grams of sulfathiazole were inserted. The anal sphincter was dilated. Colostomy was not thought necessary. The pathologist reported an inflammatory fistulous tract in the tissue from the abdominal wall, cystic oophoritis with multiple small tarry cysts in the ovary, probably endometriosis and endometriosis of the sigmoid.

The hospital course was uneventful except for a possible early bronchopneumonia on the seventh to the ninth postoperative days, which completely cleared up.

Case 3. The patient was a married woman, age thirty-six years, who had had two children. She was admitted to the clinic in December, 1934. Two weeks before she had had an exploratory operation for dysmenorrhea. At that time the abdomen was closed because she was thought to have an inoperable malignant lesion. On examination at the clinic a large cervical fibroid and firm induration of the pelvis were noted.

On opening the abdomen at operation on December 31, 1934, extensive endometriosis of the entire pelvis and bilateral hemorrhagic cysts of the ovaries, each 5 cm. in diameter, were found. The sigmoid was intimately attached to both ovaries and the fundus of the uterus. The cervix was split to deliver a large cervical fibroid. Total hysterectomy and bilateral salpingo-oophorectomy were performed.

The patient returned two years later with an adenoma of the thyroid and hyperthyroidism. There were no symptoms referable to the pelvis, and the pelvic examination gave negative results.

Case 4. The patient, a married woman, aged thirty-four years, who had had three children, was admitted to the clinic in April, 1939, because of dysuria and pain in the vagina during menses. Five years before admission suspension operation on the uterus and sterilization had been carried out. On examination at the clinic the uterus was enlarged and fibrous and there was a firm mass beneath the bladder. The vaginal mucosa showed some dimpling anterior to the cervix. On cystoscopic examination the trigone of the bladder was found to be markedly elevated from extravesical pressure. The bladder wall was negative. The patient elected to go home and had dilatation and curettage of the uterus done there. In May, 1940, she returned because of increased dysuria with the menses and frequency at that time. There was a feeling of constriction in the bladder and menorrhagia had developed.

At operation adenomyomas were removed from under the base of the bladder. The fundus of the uterus which was adherent to the bladder was freed and total hysterectomy was performed. The ovaries and tubes were also adherent and were removed.

Six months later the patient returned because of severe abdominal pain similar to her previous menstrual pain. Cystoscopic examination revealed several regions of petechial hemorrhages in the vesical mucosa, and there was a mass with nodular concretions beneath the base of the bladder. A castrating dose of roentgen rays was given, and two years later the pelvis and bladder were negative.

In this case probably some ovarian tissue was left at the time of operation.

Case 5. The patient was a married woman, aged forty-seven years, who had had two children. Eleven years before examination at the clinic she had undergone dilatation and curettage, suspension of the uterus and appendectomy.

On admission to the clinic the patient complained of pain and fullness in the rectum and vagina which followed immediately the menstrual periods and had been present for one year. She thought she could feel a small growth in the vagina. Metrorrhagia had been present for six months.

Pelvic examination revealed a hard tumor, 4 by 3 cm., high in the rectovaginal septum. On proctoscopic examination the rectal mucosa of the anterior wall of the bowel, 8 cm. above the anus, was found secondarily involved in the lesion in the rectovaginal septum. Motion was limited, and there was no obstruction.

Biopsy in January, 1927, revealed an adenomyoma of the rectovaginal septum. A menopausal dose of radium was given. Eleven years later the patient wrote us that she was well.

Case 6. The patient, a married woman, aged forty-one years, who had had two children had the round ligaments shortened five years and one year before admission to the clinic. She came because of an abdominal tumor which had appeared eight months after the first operation and had been painful with menses for three years. A nodular ridge of tissue in the suprapubic region extended laterally parallel to the inguinal ligaments.

At operation in February, 1928, an adenomyoma measuring 10 by 6 by 3 cm. was excised. The mass was lying at the point where the round ligaments had been brought through the inguinal rings. Radium was applied to the abdominal wall. Ten years later she wrote that she was well.

SURGICAL PROCEDURES OR OTHER TREATMENT

Many of the operations for endometriosis are difficult as a result of the firm adhesions and considerable destruction or invasion of the pelvic peritoneum. In many cases on attempting to free up the cystic mass a ureter is exposed or is freed up along with the tumor and unless special care is exercised it is likely to be injured. Injury to the sigmoid or bladder is also a real danger.

When both ovaries are involved extensively, no matter what the age of the patient is, it is advisable to remove all ovarian tissue, the uterus and both tubes. When only one ovary is involved and the patient is less than thirty-eight years of age, I believe it is advisable or at least justifiable to remove the involved ovary and the tube on the same side and to save the uterus, the normal ovary and tube, especially if the patient is interested in childbearing. It must be remembered, however, that the patient may have later trouble and require complete castration either by operative procedures or radiotherapy. If this is necessary, menopausal symptoms can be controlled in a large measure by using stilbestrol or theelin.

From the gross appearance of endometrioma of the bowel a diagnosis of carcinoma is frequently made and radical operations are performed. For young women who are good surgical risks, I believe this is often preferable to removing all ovarian tissue but it must always be kept in mind that complete removal of all ovarian tissue or a menopausal dose of roentgen-rays or radium will stop growth. In fact, such treatment will cause many of these tumors to disappear completely and is relatively safe, whereas resection of the pelvic portion of the colon must always be regarded as a major operation with a definite risk to life.

Extensive ovarian involvement with large chocolate cysts and extensive pelvic implants and adhesions often make conservative operations difficult and in such cases total abdominal hysterectomy with removal of both tubes and ovaries is generally indicated. In younger women when ovarian tissue and the uterus are saved and dysmenorrhea has been a real complaint, presacral neurectomy is advisable.

Moore, Herring and McCannel reviewed twenty-seven cases in which a diagnosis of endometrioma of the bladder was made. They advised local excision for young women and castration either by radiotherapy or operation for women close to the menopausal age and when symptoms were slight and the menopause close, they advised waiting in the hope that they would pass through the menopause and thus trouble would be averted. They stated that local treatment by fulguration is not indicated. In this series from the clinic there were fifty-two in which endometrioma involving the bladder or bladder peritoneum and six cases invading a ureter.

In the great majority of cases in which an operative diagnosis of endometriosis is made, it is an incidental finding in association with other pelvic lesions, most frequently with fibromyomas of the uterus. On the other hand, many patients come to operation as the direct result of extensive ectopic endometrial growths which have been correctly diagnosed before operation. In many cases such growths may involve not only the uterus and ovaries but also multiple implants of various size, from a pin point to 1 to 2 cm. in diameter, will be found on both the visceral and parietal peritoneum of the pelvis with extension in many cases to underlying structures.

All hemorrhagic cysts of the ovaries are not due to endometrial tissue but most of them, at least the ones with tarry material in them, undoubtedly are and if enough sections are taken and carefully examined endometrium-like tissue will be found in most of them. If any of the endometrial tissue is left along with some ovarian tissue, other tumors will develop. In case of diffuse adenomyosis of the uterus and no other evidence of endometriosis, in a woman less than forty years of age, I believe total abdominal hysterectomy with preservation of one ovary is advisable.

In 115 cases in the series at the Mayo Clinc endometrial growths in the sigmoid, rectosigmoid, rectum or rectovaginal septum produced indications for operations and in thirty-six of these 115 cases some previous operation had been performed. The outstanding symptoms in all these cases was dysmenorrhea, menorrhagia, metrorrhagia, constipation, rectal pain and rectal bleeding. Obstruction was noted in only 6.3 per cent of these 115 cases. In four of the cases definite signs of obstruction were present and in two colostomy had been performed five and six months respectively before admission to the clinic. In one case rectovaginal fistula followed an operation for a tumor of the rectovaginal septum elsewhere and in at least nine cases a diagnosis of malignancy had been made. In treatment at the clinic, resection of the large intestine was thought advisable with either an end to end or an end to side anastomosis in fifteen cases and in seven other cases, a portion of the anterior wall of the sigmoid was excised.

In twenty-six cases endometrial growths were present on the visceral peritoneum of the small bowel. In one case of definite obstruction resection was performed.

Besides the cases already referred to, which necessitated major operations for their correction, many unusual cases of adenomyoma have been noted in this series. In thirty-two cases endometriomas were found on the anterior abdominal wall. Most of these followed previous operations on the uterus, tubes or ovaries and can be accounted for by the transplanting of endometrial tissue into the abdominal scar at the first operation with subsequent growth. Some cases, more difficult to explain, however, have been reported in which the only previous operative procedure had been on such organs as the gallbladder or appendix. One patient who had a second recurrence of an endometrioma in an abdominal scar following a caesarean section committed suicide, no doubt thinking her condition was malignant.

Another interesting group of cases was those in which endometrioma occurred in the umbilicus and the condition is hard to account for except on a serosal theory or theory of metaplasia. In these cases as well as others in which the condition occurs in the inguinal canal, labia, and so forth, no special treatment is required except thorough surgical removal of the growth. If any endometrial tissue is left, the growths will continue to develop until the patient reaches the age of menopause when they gradually disappear.

SUMMARY

The genesis of adenomyomas or endometriosis is still an unsettled question. Probably some tumors containing tissues resembling the endometrium are in no way related to the mullerian ducts. Adenomyomas are of special interest because they represent a nonmalignant invasion of normal tissue and such a condition is not recognized by pathologists as occurring anywhere else in the body.

It is recognized that many adenomyomas are found accidentally at operation and apparently do not cause symptoms. In other cases they may cause symptoms or may endanger the life of the patient.

In some cases removal of the tumor is indicated, but in other cases bilateral oophorectomy or use of radium or roentgen rays is a safer and quite as satisfactory a method of controlling the condition.

From January 1, 1923, to September 30, 1943, a diagnosis of endometriosis has been made from tissue removed in 2,062 cases at the Mayo Clinic.

ASSEMBLY DINNER

Thursday Evening, October 28, 1943

7:00 P. M. — PALMER HOUSE

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Chairman, Chicago Executive Committee, and Annual Dinner Chairman

OSCAR HAWKINSON, M. D.
President, Chicago Medical Society, Chicago, Illinois;
General Chairman, Chicago Assembly

THE CHAIRMAN: Before coming in here this evening, ladies and gentlemen, I was carefully instructed that I shouldn't talk too much. It made me think of Ole Olsen. (This story is older than the oldest inhabitant, probably, but I think some of the older men may enjoy it because it will bring back memories of other days.) Ole took his sweetheart for a buggy ride, and as they were driving down the lane the horse was well trained, not requiring much attention, evening was falling, the moon rising from the East, the whippoorwills singing their evening song, the apple blossoms heavy in the air, and it was spring, and Ole suddenly said, "Olga, will you marry me?" And she said, "Sure I'll marry you."

They drove on in silence for quite a while and pretty soon she said, "Ole, what's the matter, you no talk to me?"

"Vell," he said, "Ay tank Ay talk too much already." (Laughter)

The Interstate Postgraduate Medical Association of North America contributes a great and valuable service to our medical men. As its President, this organization selects one whose dignity is commensurate with its own achievements. Nothing that I may say tonight or that anyone might say of Dr. Lahey could add one cubit to his fame or stature. In the field of medicine he is preeminent, a great teacher and a great surgeon; modest in his great attainments, kindly and gentle in manner. He has for years given his time and counsel to organized medicine, one year ago having retired from the presidency of the American Medical Association and now he is serving our country as Chief of Procurement and Assignment of Physicians. In every way he is one whom we all delight to honor, and so, ladies and gentlemen, I have the great pleasure and privilege of welcoming and presenting to you the President of the Interstate Postgraduate Medical Association of North America, Dr. Frank Lahey, who will speak to you on Some of the Medical Problems of the War. (Applause)

SOME OF THE MEDICAL PROBLEMS OF THE WAR

DR. FRANK H. LAHEY: Mr. Chairman, Ladies and Gentlemen: First, in behalf of the Assembly, I would like to say that this body is much indebted to the physicians and other citizens of Chicago who have contributed so generously of their time and efforts in the arrangements for this meeting. The Toastmaster said that little preparation was necessary, but all of us who have participated in these events know that they require organization. So we do wish to express to them our sincere thanks and appreciation.

Dr. Sullivan spoke to me last year concerning the advisability of having this meeting this year, and I said then, and I say again now, that particularly during a war we need continuing medical education. In postgraduate education, this group plays as great a part, if not the greatest, of any organization in this country. These meetings are not in any way in opposition to the war effort, but rather a real contribution. If education of men in the care of the wounded is necessary, then so is also the continued education of men in the care of civilians.

The position of the Central Board of the Procurement and Assignment Service is an extremely difficult one. It is a serious obligation to be charged with the responsibility of trying to determine what is a just apportionment to the armed forces and civilian population of a nonexpansible commodity such as medical personnel. Add to that the responsibility for dental, veterinary and sanitary engineering personnel, and it becomes—has always been—a far from simple undertaking.

Furthermore, I am certain that the efforts of the Procurement and Assignment Service will never be fully appreciated, and I speak not only of the Board but also of the men in Services of Command, state chairmen, and most important, in the local committees, who have done so much in determining essentiality and availability. These men, who have contributed so much time, have received very little credit, and I wish to express to them great appreciation on the part of the Board for what they have done. Also, let me say that after having lived with the Central Board from the very beginning of the war, it is a most satisfactory group of men, each possessing attributes and qualities peculiar to himself, and each, I believe, very valuable. The members have been understanding and cooperative, and have assumed their obligations most satisfactorily.

You are all participants in the program of medical, dental, veterinary, sanitary engineering and nursing contribution to the war effort, whether you are physicians, physicians' wives or laymen. Consequently it seems to me that I should report to you on the present status of medical personnel and its problems. We should survey the situation in terms of our resources in medical personnel, how the physicians have been apportioned, how many remain and how they should be distributed.

There are in the Army today a minimum of 40,000 physicians. I talked with Major Luth this afternoon, and very probably there are 41,400, safely 40,000; and in the Navy, 10,100 physicians, counting 300 in the Coast Guard.

There are 47,334 physicians in the country under 38 years of age. If we eliminate those who would be disqualified physically, based on our experience tables to date, we would disqualify approximately 12,831. This would leave physically qualified and acceptable to the armed forces, 34,503 physicians under 38. There are in the service under 38, 30,130 physicians. There remain, on the basis of experience table of rejections, 4,373 physically acceptable physicians under 38 years of age.

The number between 38 and 45 years of age is 28,381. On the same prediction tables of disqualified men, we would disqualify 8,519, which would leave acceptable for the service, 19,862 between 38 and 45. There are in the service between these age groups 8,788, leaving available and qualified, 11,074. Combining those under 38 years of age and between 38 and 45, we have 15,447 physically acceptable physicians under 45.

We have to remember that of all the physicians in the armed services coming from civil life, there are only 5.2 per cent over 45 years of age, represented by 2,800 out of a total of 50,000 to 51,000 physicians. Of the approximately 15,000 under 45 available on the basis of physical acceptability, very probably 7,000 are in states over their quotas and classified as essential. Consequently, excluding these, there are approximately 10,000 physically qualified physicians under 45. Out of this group must come some of the essential teachers in medical schools, which today are devoted almost entirely to the production of medical officers for the services. Out of this group must come also the pathologists and those men necessary for industry. After having visited the Kaiser and some of the other shipbuilding plants, I have realized that physicians who in case of accident must go down in the holds of the ship, who must go over the building material and around the cranes, must be within the active age group.

It is apparent, therefore (and I think it is one of the problems we all face), that we have passed the stage where the situation is easy to handle from either the civilian or military point of view. We have to remember that of the available physicians under 45 years of age, all but 15,000 physically acceptable are in the services, and that 120,000,000 people literally are being cared for by 57,000 physicians between the ages of 45 and 65 and 28,000 physicians over 65 years of age. The 28,000 physicians over 65 cannot be considered more than 33 per cent useful. We, therefore, have about 65,000 physicians up to 65 caring for 120,000,000 people, as opposed to 50,000 to 51,000 physicians caring for about 10,000,000 in the armed forces.

That is a reasonably accurate presentation of the situation today, and what will happen beyond this I do not know.

We must also have in mind a practical picture of what the situation is in replacements. The yearly production of physicians under the accelerated program is approximately 7,300 physicians a year. Up to 1945, which is the first year when 20 per cent of the places in medical schools are reserved for women and physically disqualified men who will defray their own expenses and who will provide replacements for civilian care, civilians will get only rejectees, amounting to not over 700 physicians a year. We have estimated in Washington that up to 1945 not more than 10 per cent of the graduating classes will be obtained for civilian care. From 1945 on, I have grave doubts that any medical school can fill its reserve 20 per cent quota of vacancies with women and physically rejected men willing to defray their own expenses in the presence of the other 80 per cent of the student population in uniform with tuition and maintenance paid by the government plus $50 a month reimbursement. I doubt very much, and the deans of the medical schools agree, that we can count on over 1,000 physicians a year for civilian care under this plan.

This brings us to a fact with which we are all familiar,—that we have had a yearly depletion by retirement, death and disability of 3,800 physicians, which according to recent investigations by the American Medical Association is increasing. The number of physicians dying in 1943 so far is well over the number in 1942 and in 1941. Therefore, in addition to the 700 to 1,000 from medical schools, plus what we can recover out of the yearly discharges from the Army and the Navy, we probably have not over 1,800 a year in terms of our experience to date. Out of this number, perhaps 800 are discharged for reasons which would make them unacceptable and unavailable for civilian care. Most optimistically, we probably cannot count on over 1,500 to 2,000 physicians a year from all sources to replace the 3,800 being lost each year.

We have a majority of the physicians under 45 years of age in the armed services, and the physicians largely over 45 years of age caring for civilians. I do not mean to make the comparison at all odious, but a large number of those under 45 in military practice are not being worked to their full capacity, or cannot be, while the ones over 45 are mostly being worked up to and, in many instances, beyond their capacity. In addition, at least 5,000 to 6,000 physically fit physicians are being turned over to the armed forces yearly to take care of replacements. On the other hand, there are around 65,000 physicians growing older each year, working harder than they have ever worked to take care of 120,000,000 people, and with a yearly replacement supply which probably does not meet the needs by 50 per cent.

I do not mean in any way to make this comparison to the detriment of the armed forces, but to present an honest, over-all picture of what the situation is and to call attention to the fact that it will get worse yearly. That is the problem we have to face, and when I say that we have passed the stage of surpluses, here is the evidence to prove it! We must, I believe, be less carefree in both civilian life and the armed forces in the utilization of physicians.

There is another thing about which we are all concerned, and that is the so-called 9-9-9 program. We asked the Surgeons General to let us have one-half of all the physicians after they had completed their nine months' officership to continue in junior residency, and of that number one half for the next nine months in senior residency. They did not feel they could meet the first part of the program. They compromised with nine months' house officership for all medical graduates, one third of the nine months' graduates to be deferred for another nine months as junior residents and one half of that one third for another nine months as senior residents.

We all have seen depletions in hospitals in house officers and residents, and it will require great understanding and cooperation to make this program work. It will, however, I believe, work more justly for the Army, the Navy and civilians than any other program that could be designed, and it has been accepted by the Procurement and Assignment Service and the Surgeons General.

We have another problem, a most difficult one for me to face, and when I refer to myself I mean the Central Board. What is the point at which civilian care is endangered? It is not easy to obtain the information with which to be just in answering this question. Making this decision is a thankless job at the best, and one which will occasion criticism no matter what position is taken.

Today the Navy is suffering more than the Army from lack of physicians. The Navy is expanding more rapidly than the Army, and on the basis of the 10,000 physicians, the Navy is under 4 per 1,000 (about 3.8 or 3.9 per 1,000). The Army, on the basis of 40,000 physicians to 7,500,000 troop strength, is on about a 5 plus per 1,000 basis. I do not know whether they can go on with that supply of physicians, but I do know that I would like to see every physician who has been classified as available in these United States compelled by one method or another to have a physical examination to find out whether he is acceptable, and if he is acceptable, take a commission. I would like to see the Navy get at least 3,000 more physicians, and I would like to see the Army, if possible, get 2,000 to 3,000 more, and more if we can spare them.

Each of you can be of great service at this time. The physicians of the United States are divided into those who have been classified as essential and those who have been classified as available. The ones who are essential should be absolutely conscience clear because they have been so classified not by themselves but by uninfluenced persons. There have been mistakes and, I have no doubt, some prejudices. There has probably been some politics, but these situations occur even perhaps under Selective Service. In general, in my opinion, the classification of physicians has been as fair and as just as Selective Service, if not more so. The plan is the same. If the physician is classified as available and he does not like it, he can appeal to his state chairman. If the verdict is unsatisfactory, he can appeal to the Corps Area chairman, and after that, to the Central Board.

If you have been classified as essential—and this is the thing for you to take home to your committees—

and you want to go into the service, you have an obligation to find a replacement. Some men have taken the position that it is the duty of the Procurement and Assignment Service to find them a replacement. However, no one is better able to handle this situation than the man who wants to be replaced. He and his state group know better than anyone else who could possibly replace him, and, I believe, if he will work with his state group he can in most instances find a replacement.

If, on the other hand, a physician is classified as available—and this is where you can help us get this last group of men who are reluctant to go into the service—encourage him to take the physical examination to find out whether he is qualified, and if he is, to seek a commission.

A physician who is classified as available has one outstanding obligation. If he does not believe he is available he should question his classification, as does the man in the draft. He may appeal to his state chairman, his Corp Area chairman, and the Central Board. If he has not questioned his classification he must admit that he shows a lamentable lack of interest in his country's need. If he has questioned his classification, has not been sustained and has then not sought a commission, it is the privilege and duty of those in his community to know that he has set himself up as judge, jury and advocate in his own case.

Publicity is a dangerous weapon for us to utilize. We would accomplish great injustices by the use of published lists because of the changing status of physicians who are classified as available, have sought commissions and have been rejected. But if you will inquire of the men of proper age not in the service whether they have questioned their classification, and if they have not, why they have not sought commissions, you can, I believe, thus legitimately bring pressure upon them.

We can, of course, finally utilize the draft. The draft already is available up to 38, and I doubt very much if by any legal process one can select a special group such as physicians and make it function up to 45 years of age. I would be perfectly willing to have the draft age for physicians raised to 45 if we could retain what I think is so important, and that is that only those be drafted who are classified as available by the Procurement and Assignment Service. However, I doubt whether this could be done legally.

Because of the Board's serious obligation to the armed forces, as well as its relationship to the civilian population, it is in a particularly peculiar position regarding questioning the Army and the Navy as to their efficient utilization of physicians. There is not the slightest likelihood that any man on the Board seriously will consider himself able to settle problems in terms of being a military expert. The Board will, as its duty, however, continue to question Army and Navy decisions and distribution and utilization of physicians at home—not on the fronts. If the fronts have a surplus, they are welcome to it because who knows what is developing and who knows what they may need! This problem belongs to the Army, but at home it will continue to be the duty of the Board to question medical decisions, distributions and utilizations in the armed forces. And it will always do so reasonably, always in the bosom of the family, so to speak, and never in the newspapers or critically before public lay audiences.

The Army and the Navy likewise must have the right to question, investigate and suggest changes in civilian medical care. Those responsible for medical care in the Army and the Navy have the most serious obligation of providing the best medical care for the men in the services. This task is of such magnitude as completely to occupy them, and does not give them an opportunity to become acquainted with the status of civilian and industrial medical care, medical schools and hospital problems. We, too, know all too little as to the details of military needs, plans and efficiency of utilization. I am certain that this is one of the problems that will present itself in the future as a possibility of improvement on both sides in the better management of medical distributions between the civilian population and the armed forces.

Having lived with this problem since the beginning of the war, I know that the surplus period of medical care in and out of the services has passed and that from now on the situation will be more complicated for both sides, and will require closer cooperation, understanding and compromises. With civilian care being delivered largely by physicians between 45 and 75 years of age, working harder as a group than ever before, together with the physical rejectees under 45, the number each year lessening because of inadequate replacements, think how difficult the problem can become!

In relation to this, we must also remember that thing which is always a vague threat but nevertheless has to be considered, and that is the possibility of a civilian epidemic. On the other hand, we must be equally just and realize the increasing problems of the Army and the Navy, with increasing personnel, possibly increasing casualties, the need of extra men on the fronts, and also the possibility of epidemics. This is a little balanced, however, by the fact that the Army possesses only 5.2 per cent physicians over 45 years of age, and have an age group capable of carrying heavier loads than the group of physicians supplying civilian care.

We must, I believe, begin to meet the pressure by improvising, by doing some things we would not have done when we had such surpluses. We can stretch civilian care more if necessary and improve civilian utilization of physicians. There must be no so-called luxury medicine. Physicians must work longer and more efficiently. Operating staffs must alternate, some working in the morning and some in the afternoon. With less in the way of operating room equipment and facilities, we must not let what we have rest idle part of the day, and the same is true of x-ray and clinical laboratories.

The conserving of an unexpansible commodity such as physicians involves keeping the young, vigorous men completely occupied in their profession just as it involves the thrifty civilian use of the medical personnel between 45 and 75 years of age. I doubt if we can work most of the older men in civilian life any harder, since those who are not working at peak capacity are few. I am sure there are more who could do obstetrics. There are some who are taking advantage of their age not to do night work, but they are limited in number. In general, I think the older men are working hard and doing an excellent job.

Another problem which we have to face is that of nursing. When one realizes that our cadet nursing program involves 62,000 to 65,000 nurses, that we have around 40,000 nurses in the service now, and that the Army is asking for 2,000 a month and the Navy 500 a month, one also appreciates that nursing care must be proportioned. A special nurse in our hospitals must look after 5, 6 or 7 patients. There can no longer be eight-hour nurses who are literally companions for the well-to-do patients who can afford them. We must utilize the older nurses to better advantage.

Added to these are other problems! Do you realize that approximately 2,000 patients per month are being turned over to the Veterans Bureau and that very soon this will involve a problem of medical care? Do you realize that the Navy has 60,000 beds and will increase up to 80,000, and that the Army has 375,000 in this country and 120,000 abroad, and will increase 100,000 to 200,000?

We, therefore, have to think in terms of what the armed forces could consider as improvisations, as utilized by some of our Allies. We have to consider at least, and to suggest, the possibility of interchange of service between civilian and medical agencies. We have to consider in this country where we have so many vigorous men literally within compounds carefully fed, housed and guarded, the possibility of a lessened number of physicians to care for them, and the possibility of medical pools and rapid movement of physicians if necessary by air to meet emergencies.

We have to consider the more complete utilization of medical personnel for medical purposes. As I see the hospitals of the Army and the Navy, I am appalled by the amount of time consumed by medical men in keeping records. No man who runs a clinic would consider for a minute paying for medical time to be utilized in doing clerical work. Consequently we have to consider the provision of secretarial aid through either civilian help or the services.

Not today or in the coming year or years can the civilian or the military medical authorities say that it cannot be done. There is nothing more distressing to me in an emergency such as this than the words, "It cannot be done." That attitude defies all new plans. If ever we needed to avoid irritating controversies and to seek tolerant understanding, it will be in the coming year or years as we endeavor to meet a situation which will become more and more trying not only to relation to our intellectual capacities but in relation to our nervous systems and our ability to adjust ourselves understandingly to each other's problems. (Applause)

THE CHAIRMAN: The next speaker on the program will talk to us on Some Observations on Combat Surgery, Brigadier-General Fred W. Rankin, Lexington, Kentucky; Consulting Surgeon, Office of the Surgeon General, United States Army, Washington, D. C.; Immediate Past President, American Medical Association; President-Elect, Interstate Postgraduate Medical Association of North America. I should like to introduce General Rankin. (Applause)

SOME OBSERVATIONS ON COMBAT SURGERY

BRIGADIER GENERAL FRED W. RANKIN: Mr. Toastmaster, Ladies and Gentlemen: Early in July I received a detail which carried me around a considerable part of the world and enabled me to visit not only all of the theaters of operation where we have troop installations, but to visit with and listen to many of the higher governmental people of the various allied countries. It was a trip which, since my duties were not entirely those of a medical inspector, permitted me to see many phases of the operations and occasionally to see governments in operation in this global war. I know no reason why I should not tell you the route which this party took, because we traveled entirely by air, and it was a very interesting and well planned route, I think, going from the United States first to Newfoundland, then to Iceland, England, on over into the active theaters in Africa and around that region.

Iceland is a bleak island, where the winds get as high as 130 miles per hour, where it is always dusty, where there is little grass, where there is little vegetation of any kind, where there are long nights and where the temperatures get extremely low.

We had at one time some three or more divisions of troops in Iceland and it was an important strategic point. As a matter of fact, unless the British had moved in unceremoniously it is entirely possible that the Germans might have done so earlier in the war. When we supplemented the British garrison and took over the defense of the island, we put in a large number of troops, about three divisions or more, as I said. Since the war has moved away, plans for increasing this garrison enormously have been revised and fewer troops are there at present.

I saw people who had been in Iceland twenty-three months, and since this was in the earlier part of my journey I did not accept so eagerly the suggestion that a policy of rotation in foreign service should be adopted as soon as practical. As I went further and further around I became more and more converted to that opinion and I am sure that the War Department feels that where at all possible some such policy should be instituted.

From Iceland we went to England and there I saw a huge army and a huge air force of British and American allies in preparation for whatever lies before. Britain has withstood the war for four years, has withstood the horrible blitz of which you all know, and I must say with characteristic British courage has eradicated many of the scars in London which were made by the blitz of August, September and October of '41. Our combat divisions are going through a process of training there, but our air divisions are fighting. We have a large air force, the 8th Air Force under Lieutenant General Ira Eaker, and it was my pleasure to spend three days with the 8th Air Force and go through many of the installations, to be present at General Eaker's conferences, to visit the bomber command, the fighter command, and to listen to the overall plan which they are carrying out daily over Germany. I know of no more inspiring spectacle and nothing that could make one feel more humble than to stand in the presence of a bomber crew or bomber crews, as I did, who were being briefed to take off that night for missions over Germany. Many of those men didn't come back the next morning. That goes on from day to day; it goes on in the British Air Force similarly. These airmen are doing to the Germans exactly what your newspapers tell you they are doing. I saw the pictures of reconnaissance over Hamburg after Hamburg had been blitzed; I saw the pictures of Cassel, of St. Nazaire, and many, many other towns that had been attacked by the RAF and the American 8th Air Force. As you know, our Air Force bomb by what they call precision bombing; that is, they can hit their targets within a circle of a thousand yards from a 20 to 25 thousand foot level, and they do it and bring back the pictures to show it, and they go back the next day and take more pictures to see what the final result has been. The British, on the other hand, indulge in area bombing; that is scattering bombs over the entire city or the area where we have done precision bombing the day before. They do the night bombing, and that is good for the German soul, too, because he loves to come over and bomb women and children and towns, and I think maybe he understands a little better the technic of the blitz now that he has had to suffer a bit of it himself.

Those people are doing a magnificent job. One cannot say too much for the air forces of the two nations. I don't think you can be as enthusiastic, particularly not if you are a doughboy at heart, as the air people are about winning this war singlehanded; they seem to be infected with a peculiar virus which gives them an enthusiasm and an optimism which so far as I am concerned I hope is realized, because they are thoroughly convinced that given a little time they can win it singlehanded. More power to them, I say, and so do all other men who serve with the ground forces. But I have a feeling that when the final answer is made, when the final bell is rung, that the old foot soldier will be there on the ground to take over and hold the land, just as he has done in all the wars of the past.

Our medical services in Britain are in the charge of Brigadier General Paul Hawley. He may be known to many of you medical officers here, and I can assure you that our medical affairs are in able hands. General Hawley has been over there now, I presume, since before the war started. His job has been planning and training, and he has planned well, I am sure, and has established a liaison with the British which unquestionably is most useful and as the campaign progresses I am sure will be more and more advantageous to both sides. He is looked upon not only by the Army officers of both armies but by the governmental officials as a most efficient, able, and cooperative officer, and on two occasions American Ambassador Winant mentioned to me the many excellent qualities of this officer which had made him most useful not only to the Army but to the Government as well.

I was privileged to interview the great Churchill for a few moments. I say to interview—that is wrong. You go into 10 Downing Street, you are introduced to Mr. Churchill and Mr. Churchill characteristically does most of the talking; an enormously interesting person; a large, heavy, elderly gentleman, with bald head, sharp blue eyes, pink skin. Some lady said to him, "Why, Mr. Churchill, you look like my baby!"

He said, "All babies look like me, Madam," and it is perfectly true. He smokes these long cigars and his mind still scintillates. He gives one the impression of great power and intellect and I think that I am not guilty of overstatement when I say that Mr. Churchill knows what he wants and has an idea of how to go about getting it.

From England I went to where the war was going on; that is, down into Africa. The Sicilian campaign was just being wound up at that time and we went across, flew down from Land's End to Marrakesh and then back up to Casablanca where the initial landing took place, and then proceeded by rather slow stages across the entire north of Africa. If your geographical recollection is as meager as mine was of the distances involved, you would be just as surprised as I was to find that a jump of 1200 miles didn't get you very far when you were trying to get somewhere: enormous distances, peculiar terrain, mountains on one side, sea on the other, one railroad going all the way across the country and many, many difficulties of transportation.

From Mateur up to Bizerte all the impedimenta of war still lie along the road. One is not allowed to get off the roadway more than twenty feet on either side because the land mines are still there in great profusion. Marshal Rommel is a master at defensive operation, a master of this new technic of warfare which is land mines, and his associates strewed mines all the way up to Bizerte. The Engineers have cleared them out on either side of the road, but I am confident that this cruel additional technic of warfare will cost the lives of many, many innocent civilians, not only in the next few months but perhaps for the next few years. Of all the wounds that I saw in the evacuation and field hospitals of Africa, the ones produced by land mines I thought were the most horrible and the most extensive. These mines explode, as you know, from contact; one treads on them or a jeep runs over them and they go off and blow a leg or both legs off or mangle a foot or not infrequently kill the driver. They have an additional apparatus which is the latest innovation; that is, two little mines connected. The first one goes off and throws the second one up so that it comes just level with the jeep and that is even more destructive since that explodes into a man's vitals and not into his extremities—a horrible type of warfare.

I saw many of the evacuation hospitals and the general hospitals and station hospitals in Africa, and I say to you that I have never seen a higher standard of surgery anywhere than those men are giving to the American soldier.

As one who did surgery in the last war, I frankly confess that these men are doing a much better job on the average. I think that is obvious for two or three reasons. In the first place, they are better trained; there are more of them who are well trained, at least. Many of them have had long periods of training and they are young, active and alert fellows. They are so alert and so active that they refused in the Tunisian campaign in several of our evacuation hospitals to allow the auxiliary surgical groups, which are one of my particular prides and pets, to do any more work than they had to, feeling that it was their own job to carry on in their own hospital. That is a situation which will remedy itself as time goes on and fatigue and long operating hours take place.

We have suffered many less casualties in this campaign up to the present time than was anticipated or could be calculated on any table of warfare in the past. Our casualties in Tunisia were far fewer than we anticipated. The casualties in Sicily were also fewer in number. I don't quite understand why we had fewer casualties getting into Italy, but we did.

I spent a day and a night with General Clark and went through a landing operation all the way. This was not a real landing operation; this was a practice landing operation, and at the end of it, that is when we got on the LST boats and landed and took the pillboxes and did the whole thing from start to finish, at the end of the day I had convinced myself if I had ever had a doubt of it that this was indeed a young man's war. It is no place for the middle-aged, sedentary physician, over there doing amphibious operations.

The immunization program of the Army has paid great dividends. Our soldiers had been protected from tetanus completely. We have had two cases of tetanus in our combat troops. Neither of the men had had complete courses of the toxoid. Our typhus program had been proven entirely satisfactory. Our British allies and friends were unwilling to vaccinate for typhus, accepting it as a natural sequence of war, and having been at home among the Middle Easterners, the Arabs and Indians and other people who think that a certain number of lice are good for the soul, they didn't vaccinate against typhus and had a not inconsiderable number of cases with a rather high mortality rate. Our troops were vaccinated against it and we had three mild cases in Africa and no fatalities. General Bigham said to me, "Using us as a guinea pig, you have proved the experiment."

I think that that is very satisfactory: no typhoid, no paratyphoid, and the gas gangrene that was so fatal in the last war we saw very little of. I can't give you the exact number of cases they had in Africa, but I recall a series of sixteen cases, thirteen of which I think definitely were the result of bad management. Inasmuch as the wounded were handled properly in that terrain, very little gas gangrene developed. Now, there are several reasons for this small number of gas gangrene cases and not anything we did particularly. You do not have the anaerobic bacillus, Welch, and the other things that cause gas gangrene, present in large numbers in the sands of the desert. We had more in Sicily where it is mountainous terrain. We will have more in Italy as we go farther forward, and I am sure that if and when the continent is invaded we will have a return of the old familiar figures that we had in the last war.

As you know, the Army and the Public Health Service and other agencies are now working on a toxoid against gas gangrene similar to the toxoid that we use for vaccinating against tetanus. I am particularly hopeful that that will be an early development, but I am not willing to be too optimistic about it for it is a difficult experimental and research project.

Penicillin is the one thing that I believe offers the greatest hope to gas gangrene cases at the present time. I base that entirely upon the experimental work which has been done in this country and not on any large clinical series that is available. The experimental work is phenomenal. The British are using it in their cases as prophylaxis against gas gangrene, and we have both used it in some of the cases which have developed gas gangrene. Apparently we don't know the right dosage, apparently we are still far from having it solved, but it seems to be something that offers distinct hope for that group of cases.

The general overall health picture of our Army is enormously heartening in the African and Italian area. I don't know why we haven't had some kind of an epidemic or more difficulties, because sanitation as such does not exist in Arabia and in North Africa and in India. They are the only countries that I have ever been in where the same conduit is used for water and for sewage. That is true; that is not meant lightly. I have seen it with my own eyes, and everyone who has been there can verify that assertion. There is no such thing as sanitation as we understand it in those countries, and yet we have gone along and controlled the epidemics, controlled the health problems of the troops in an enormously satisfactory way.

We don't have a great many installations in India and fortunately we hurried across. We stopped at Assam on the Persian Gulf where the great British oil refineries are and where we have an enormous installation which is getting material of all kinds, particularly air, up to the Russians. As I stepped off the plane I asked the temperature, and verified it, and it was 132 in the shade and there was no shade. When it got down to 112 everybody thought the evening was coming on and it was getting cool. It is the most horrible climate imaginable and yet those people are going on there and by utilizing the hours that are cooler and by knocking off in the middle of the day and starting again around four or five o'clock they are doing a great job and are getting all of the material that we ship over to them up to the Russians. I saw a lot of Russian fliers down there who were testing the airplanes that we sent to them and who were accepting these and flying them off back up to Russia.

On over to China, however, we had a most interesting trip. It is easy to see why the Burmese and the Chinese never get together. All you have to do is to see the terrain. The Burma Road is closed, as you know. The Burma Road is not a very big road and not a lot of stuff can get up there at best. At the present time, what supplies are getting up to General Stilwell and General Chennault and General Davidson are being flown by our own ATC, and it is a difficult and serious problem in logistics because an enormous amount of gasoline has to be burned up to get one gallon of gasoline or one shipload of material over the hump into China. That is the only way we get material up there, fly it up over the hump. The hump is a tough, mean flight. I made it twice and I don't care to make it again. We have boys who are doing it every day and sometimes they make a trip and a half a day flying supplies up to Chennault and Stilwell. You go up about 22,000 to 23,000 feet. The day we went over first it was soupy and we went to 23,000 feet. You wear oxygen masks. That day we took a new plane, a DC3, and it was an unpleasant trip all the way. Coming over to Kunming, Chennault's headquarters are here. Chennault is a great fighting man, a lean, lank, dark-skinned, black-eyed fellow who just looks like a soldier, and he is a soldier; he is unorthodox, but he is a soldier. At one time he got down as low as two planes but he kept them flying and he is still there with many more planes, I am glad to say, now, and he is enthusiastically carrying on and thoroughly confident that the Japs aren't going to dislodge him and that he can dislodge the Japs.

From Kunming we went up to Chungking where General Stilwell has his headquarters. There is a hardbitten fighting man. I stayed at his house three days, and the second morning when he addressed me in profanity which I understood I knew I'd like him. He is a very frank, outspoken man, but a serious student. His job is training the Chinese armies, largely. He hasn't a great deal of ground force or anything except service troops, and these two air squadrons, the 14th and the 10th, but he has the confidence of the Chinese, and since I left there Lord Mountbatten has been made the Commander in Chief of the C.B.I. I don't know what the relationship between the Chinese theater and the headquarters will be, but I can assure you it will one of stout cooperation and I can assure you that things in China are in good hands as long as General Stilwell has charge.

I think the highlight of the trip, at least as far as I am concerned, was a visit with the Generalissimo and Madame Chiang Kai-shek. The Generalissimo is unquestionably the strong man of China who holds the various factions together, has subsequently been made President of China, and I believe is the one hope of continued allied cooperation and support.

As for me, I subscribe to all of the adjectives the correspondents have written about the Madame. She deserves them all and I think is a most important factor in the Chinese Government.

From China we had to go back through India a bit in order to get to Australia because there were only a couple of ways we could do that and one is to retrace our steps, so we flew down to Calcutta and then over to Ceylon and from Ceylon to Australia we made the long water hop. It is some 3200 miles over water and the point of no return right in the middle is over a little island about the size of this room where there is no landing field. We made it easily in fifteen hours and forty-five minutes and, as I say, were the first land plane ever to make that hop. PBY's make it in about thirty hours, but these Liberator bombers I think will in the future make it both ways much more expeditiously.

I met General Somervell in Fiji and he was going back over the same route only in reverse. I have since learned that he made the trip but not quite as quickly as we did. We had tail winds, of course, and very favorable circumstances all along. It was a totally uninteresting trip and one had no feeling of being a pioneer at all in making it.

In Australia we saw a good many of our hospitals, both in the Australian mainland and up in the forward echelon, which then was in Port Moresby in New Guinea. General MacArthur is there; that is his headquarters. He was there at the time —that was just after the Buna-Buna campaign and Salamaua had just fallen while we were in Australia, and when we left the campaign around Lae was in progress. One cannot quite grasp by description the difficulties of jungle warfare. It is so entirely different from mechanized warfare where you have a battlefront, sketchy it is true, but you have a battlefront and you have a regiment or a battalion or a division or an army. Over in the jungle you do not have a battlefront. The whole operation is a small kind of operation, more of an individual operation from the time of the amphibious landing and it is particularly difficult from every standpoint. The greatest enemy we have is malaria. We had more casualties from malaria than we had from battle wounds among the troops who went into Guadalcanal; there is no avoiding that. Men who have been up into New Guinea are better controlled, have better malaria discipline, have better malaria preventives, better mosquito repellents and more malarial control all the way around.

I think that the medical department is doing a good job with the malaria situation. After all, it is a command job. It comes down from the top, and General MacArthur is a stern disciplinarian and he definitely is malaria conscious. I think we will have fewer and fewer malaria casualties as time goes on, as better trained troops go into the area, and as we get further forward off this particular spot where we are now which is terribly infested and difficult to control.

I think that the situation down there is progressing slowly but surely. I came away from MacArthur's headquarters with the feeling that I had been in the presence of a very great man. I would not say that one gets to know the General on a two-day acquaintance, but I would say that he was rather an austere person, and one realizes immediately that he is in *the* presence when he sees MacArthur. The man has great charm as well, and great force. He has a burning desire to go back to the Philippines, and I think that desire must be realized. I watched him talk of the Jap and talk of the Philippines, and he did it with great seriousness and a certain emotion that was very moving.

I cannot tell you how every soldier in the Southwest Pacific feels, what a hatred he has for the Japs. It doesn't take long to develop that down there and they all have it fortunately. I think that is their business, to kill Japs, to hate Japs, and I do not hold with these more mild and saintly people who would fight barbarians and savages, for that is what the Japs are, in a sportsmanlike manner. Most of them do not understand sportsmanship; they do not have any rules that our people have; they are savages and it is going to be a long and tough and hard fight down there, I am sure. But I want to say to you that when it is in the hands of men like MacArthur and Admiral Halsey and Admiral Nimitz, there need be no fear of the ultimate outcome.

I came away from this trip with a profound admiration for the work that the medical department is doing in all of the theaters of operation and with the profound conviction that no other country in the world, and I say this advisedly, gives a medical service to its armed forces which parallels that which our armed forces are receiving. I believe that is true. I furthermore came away with the greatest admiration for the military leaders not only of our own troops but of our allies, and with the firm belief that these men understand the problems that they have in hand and that they will carry them through to a successful conclusion. (Applause)

THE CHAIRMAN: I am quite sure that occasionally the men in this room, when they weren't quite so busy as they are now, have gathered in the locker room and after telling all the stories they could think of would begin talking about the old professor, someone loved and admired and respected a great deal, and after a while someone would say, "They were giants in those days."

We have actual living proof that the giants are not all dead, and I should like to introduce to you Dr. John F. Erdmann, Attending Surgeon of the New York Postgraduate Medical School. He has been one of the best instructors that this Assembly has had, one of the most popular over many years. Dr. Erdmann. (Applause)

DR. JOHN F. ERDMANN: Mr. Toastmaster, Ladies and Gentlemen: When we are asked to speak before a medical society or organization such as this one we have ample time to prepare for it. I was notified as I was coming up the stairs to enter the dining room. I am going to make it very short, because there is another speaker who is anxious to get in the traces. My reasons for coming to these meetings are more than twofold. I come here for enlightenment, instruction, to have the rust rubbed off, and to enjoy my friends in the organization.

May I say goodnight to you all, and thank you. (Applause)

THE CHAIRMAN: Dr. Lahey was obliged to leave to catch a train and I am afraid that General Rankin will be obliged to do the same thing pretty soon, and that will leave only the Toastmaster — you stay with me, Dr. Erdmann.

Everybody loves a good Britisher. It doesn't make any difference how he handles his h's or his r's, we still love a Britisher. I understand, however, there is a man over in Germany who doesn't care so much for the British, and I think his name is Hitler. However, they said that while he doesn't love the British he has some respect and a good deal of admiration for them. I am happy tonight that we have one of the bulldog breed with us, Mr. D. Graham Hutton, Director of British Information Services, London, England. He will talk to you on The United Nations in War and Peace. Mr. Hutton. (Applause)

THE UNITED NATIONS IN WAR AND PEACE

MR. D. GRAHAM HUTTON: Mr. Chairman, Ladies and Gentlemen: As I came to the speaker's table tonight the horrible realization dawned on me that I was the only layman at this table among medical speakers. The medical profession owes a very great deal throughout the world to small animals, guinea pigs, mice, and other small breeds. The equally horrible realization dawned on me that I was probably the guinea pig on this platform tonight!

Well, let me take that same simile. Medical science owes a lot to the laboratory, to the guinea pig and the mice and the men that have worked on them.

This war is, and has been, and will be the biggest social laboratory that the world has ever seen. We have discovered (I say "we" in this case, all of us, the United Nations) in four years and two months of the greatest war that humanity has ever seen, things that would have taken us twenty, thirty, perhaps even fifty years to find out at the pace of peacetime.

I am not one of those who think that war is the worst thing in human life; nor am I one of those who think that *only* evil comes out of war. Trained as an economist and as a sociologist, knowing something of the technic of modern science, I should be an idiot, an imbecile, or a fool at any rate, if I thought that out of the wars of the last thirty years no good had come. Nobody in your profession looking back thirty years could put his hand on his heart and say, "If those thirty years had been thirty years of peace we should have made exactly the same progress." Four years, 1914 to '18,—four years and two months so far and maybe a lot more,—have brought in a very short space of time a degree of technological progress which the world has never seen before. In your profession, things are being discovered in this war in a matter of months which have outdated completely what was being done a matter of months before.

I want to step aside from that medical simile now and go to a wider canvas. The whole world, oriental and occidental, what we call western industrial civilization and what we can call antique or ancient civilization, has been groaning and travailing for a matter of years now and will continue to do so for some time, even probably after the end of the actual fighting. What is the lesson that we have discovered so far in this war?

I think that there are three main divisions of that lesson. First is the technical. The United Nations were not united in this war out of love for each other. They were united by our enemies. Our enemies attacked us or forced us into this war at different stages and at periods of time which our enemies selected and which we did not. As a result of that development a large number of nations in this world have come together with different political systems, with different degrees of scientific and technical development, with different organizations of their own people, and have fused their efforts in a matter of months. That is how old it is. In your case twenty-two months, in our case four years and two months, in other nations, Russia, for example, a matter of two years and four months. They have fused not only their fighting forces on different fronts, in the air, under the water, on the water and on the land, but they have produced things which the world has never seen before.

We who read the newspapers and listen to the radio day by day get almost a sort of Pavlov conditioned reflex in thinking that these things are generally known; but they are not known, they are unprecedented. There is no precedent for anything that we see in the world today in the history of 1914-18; nothing. In this relatively short period you have seen things which twenty-four months ago nobody in this room would have dared prophesy or even think possible. You have people of one nationality fighting under a commander of another nationality, carried on ships of a third nationality, supplied by supplies from a fourth nationality, brought together by logistical experts of perhaps a fifth nationality, and serving an army composed of three or four nationalities. That has not happened before on that scale; a matter of two years ago it would have been impossible to predict; and yet that process is working out successfully.

Sometimes I think that we Anglo-Saxons in particular have a habit of thinking in peacetime that everybody else is so much smarter than we are. You know, in both of our military schools there was always a great admiration for the Prussian army. The American and British soldiers were brought up on the Prussian strategy. There was perhaps an overemphasis placed upon this great ability and ingenuity on the part of our enemies. I do not think we overemphasized their barbarity; we underemphasized it. But as far as their ingenuity was concerned I think we exaggerated it.

Take the case of General Rommel for example. General Rommel was played up in practically every newspaper of the United Nations as being a superb strategist and a brilliant tactician, but he was completely outsmarted by a relatively unknown British General, a man whose name certainly wasn't known to you in this room three years ago, and probably not known to ninety-nine per cent of the inhabitants of Great Britain, either.

So it is with the technological and technical advances that we have made in the war as all the United Nations. It was not, in the last war, the practice for the Allied Nations themselves to pool *all* their experience. The experience that we have had in Britain of the relationship between the diet of the civil population, sustained on very drastic rationing for over four years, and at the same time coupled with the most intense drive in production and work, as a laboratory experiment is not one that we undertook willingly, but one we were forced to do. Yet the results are there, and the results have been turned over to other nations in this war in so many fields already. You think, for example, of radar. Where would the British Isles have been and where would the Allies have been today if radar had not been developed just to that pitch two weeks ahead of the enemy? Two weeks, that's all! That was the margin between the Battle of Britain going to Britain and the Battle of Britain going to Germany in 1940. It was a technical advance. From that moment onward the technic of that secret was a British secret no more; it was shared by the United Nations. It is the same in medicine. It is the same even in production.

It is the same in every kind of advance that we can make among the United Nations in order to win this war.

Secondly, look at our strategy of pooled effort.

Look back twelve months tonight: October 28, 1942. Nobody knew a year ago tonight that the Americans would be in North Africa, convoyed by the British Fleet, together with the French; that in a matter of weeks General Montgomery from Egypt would have advanced 1,600 miles, the longest continuous military advance in military history so far; have joined up with the forces of General Eisenhower; and that in a matter of months after that we should have been in Sicily; and weeks after that in Italy. Yet we have all achieved this, working as a team.

Now at this point (and I am drawing to my conclusion) I want to look forward. He would be a fool who would look forward tonight and say this or that is going to happen, and I have no intention of proving I am a fool to you. In a matter of months or years this war will be won, and it will be won by us, the United Nations. Of that there is no doubt. Mr. Churchill,—who, as General Rankin said, quite truly does look like every baby born,— told your Congress, in their sovereign might assembled, and the British House of Commons, exactly the same thing a matter of weeks ago; namely, that the whole forces of the British Commonwealth would stand beside the forces of the United States until the last Jap was ground down under our heel. But when that time comes, do you in this room tonight think that our problems are going to be easier than they were during the war, or more difficult? I am one of those pessimists, as, I suppose, all economists are we haven't much soul, I think—who think that during a war that we have been forced to fight together as united nations the problems are easier to solve, the war is easier to win. When the victory is won, however, the problems of sticking together in the peace, when we no longer have to fight an enemy, are infinitely greater of solution.

So, lastly, as I see the postwar world faintly and dimly, there are a couple of outstanding problems that we have all got to face. The first one is our post-war attitude toward the war-torn world. That world will contain ex-enemies; it will contain ex-neutrals; and it will also contain ex-United Nations, —I hope not dis-United Nations, but let's call them ex-United Nations. Our new problem under that first point will be the problem of restoration. In the problem of restoration, one of the most important contributions that the war will have taught us is the results of the laboratory experiments that have been carried out on this most vast scale.

We in Britain have had to learn a great many of those lessons, and they are not easy. For instance, I as a Britisher who spent the first two years of this war over there, listened with intense interest to what Dr. Lahey was telling you tonight. His survey of your American medical professional problems I heard in Britain two and a half years ago. That was the stage that we had been forced to reach then, and of course the problem is very much more acute over there, as you would expect, by this time. The stage of 1945 that he referred to for the United States, was reached in Britain in 1942. But this laboratory result is in our hands already; and when the peace is there and the "Cease fire" has sounded, we have the results in our hands. What use are we going to make of them?

The problems of your profession; of businessmen; of laymen; demobilization of men; equally important, demobilization of machinery; and equally important, again, demobilization of plant and capital that has been solely concentrated during the war on the output of war goods, and suddenly perhaps ninety per cent of that demand vanishes overnight with the coming of peace—these problems are not unique to the United States. It will be the same in Britain, Australia, and north of the border here in Canada. It will be true of the Far East, and with Japan, our enemy. In Europe, in Germany, in Russia, with a different economic system but with the same essential problem, conversion of the war industries to some kind of rehabilitation and restoration work will be necessary. That problem can, I am sure, be solved, and best solved in the way in which we have been forced, each and all of us, to solve the problems of winning this war,—together! That is, by pooling the experience gained from the laboratory experiment; by keeping the experts together (and here I *mean* experts) like the British and the American medical men who, on whatever front they have been placed, click together because they face the same problem with the same technic, the same vocabulary, and everything else. They were medical men together. They had a common bond. They solved these problems together. Incidentally, it is remarkable in this technical age how important specialized vocabularies are. Have you ever realized that you cannot put aviators in the same room together, no matter what nationality they are, but what they understand each other right away? The same is true of every technic which has been carried far along on the road of progress.

We must do that after this war and share the results. Those of us in Britain under the blitz learned a terrific social lesson out of that blitz. We learned an enormous amount about child psychology, for example. There were millions of evacuated children from different homes. I cannot go into the details. But when a nation like that, with 46 million people, is suddenly and completely uprooted, its whole family life broken up, its whole dietetic structure altered by force, those things have an enormous result. You don't read these things in newspapers, because they just are not news. But to you people in this room the results of those experiments in Britain would be news. They might have some medical relevance over here. Your experience over here or in the Southwest Pacific or in North Africa, together with ours, will have a very great effect upon the reshaping of the postwar world as a fit and proper place for humanity to live in.

The second and last problem that we shall have to face is, of course, the political problem in the world. (For politics I go back to the good old definition of Aristotle 2,400 years ago: Man is a political animal, an animal who lives in societies.) The political problem is one of so many societies at different levels of development, whole nations or countries, trying to live together without descending into this chaos again within my lifetime. Just as a small personal aside let me say that I, who, I confess, am forty years of age, have twice in my lifetime had to take to the cellars against enemy action in my own home city. Sometimes I think forty years of age is not very old (until my wife reminds me that it is). But that has happened twice in my lifetime. At the rate the world is shrinking and being shrunk by us (we are the people responsible, our own United Nations have contributed most of this development, the airplane, and all the other wonderful things of science that have been carried forward in the last ten years), forcing ourselves to live cheek by jowl and elbow by elbow, closer and closer, we shall have to devise the means by which we do not jostle each other and descend into another fracas like this.

I think it can be done. It is not my business, nor even is it within my ability, to tell you how it can be done. But I have learned one lesson from close association in four years and two months of war with many allies, and that is this: if you have one consuming and overriding purpose, namely, the winning of the war, it is amazing how many differences you are prepared to sink to achieve that end. My great concern,—and I think it is the great concern of everyone who stands aside for a little and looks more than, let's say, five or ten years ahead,—is that the marvelous teamwork that our technicians and our experts and our military men and the rest of them have done among all our United Nations during the war, which is slowly and surely bringing us certain victory, should not be thrown away after the war, and we have it all to do over again.

Thank you very much. (Applause)

THE CHAIRMAN: On behalf of the Assembly I should like to say that we are highly gratified with the attendance and with the continued interest you have shown tonight.

The Assembly Dinner was adjourned at 10:40 P. M.

DIAGNOSTIC SURGICAL CLINIC

Tumors of the Neck other than Thyroid

By RAYMOND W. McNEALY, M. D.
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Most physicians and surgeons are familiar with the problems which arise from the thyroid gland or goiter. They are, however, much less familiar with certain tumors, cysts and fistulae of the neck which are occasionally seen.

Lateral and median cysts of congenital origin present some of the most confusing problems of diagnosis and treatment which the surgeon is called upon to solve. Further confusion frequently arises from the tendency of these cystic swellings to form sinuses and fistulae. Mixed parotid gland tumors are not uncommon, but there appears to be some difference of opinion as to the proper treatment for them when they are diagnosed. Histologically parotid tumors have been the subject of extensive investigations.

Conclusions as to the classification of the nature and genesis of median and lateral cysts and fistulae have undergone many changes as the result of continued investigation of embryologic formations and exhaustive histologic study. Considerable difference of opinion, particularly in regard to the origin of lateral cysts and fistulae, are still to be found in the literature on the subject.

It seems to me that we must recognize that these cystic swellings may originate from many different tissues and in many instances their exact classification can only be made after a microscopic study of the cyst wall and its contents. It may be helpful also to examine histologically the structures adherent or adjacent to the cyst or fistula, since these may give a clue to the nature of the contiguous tissues.

Lateral Cysts and Fistulae

Embryologic Development. The term most commonly applied to lateral cystic tumors has been "branchial cysts." The word "branchial" is of Greek origin (*Bpáyxia*), meaning gills. There has been some tendency of late to question the use of the term "branchial cysts" except in describing those occurring above the level of the hyoid bone. The contributions of Wenglowski on the embryology of the area in question are of much interest. He concluded that the branchial apparatus cannot leave remnants in the neck below the level of the hyoid bone. The so-called "branchial" cyst or fistula below that level is regarded by him as a persistent thymic strand which originates from the third pharyngeal pouch, whence it pursues a long, oblique course down the neck to reach the anterior superior mediastinum.

While there is much merit to this critical analysis of the true embryologic origin, nevertheless it seems that one might use the term "branchiogenetic cysts," assuming that in the broad sense this would convey the general site of origin.

The following slides will illustrate how the structures of the face and neck originate from the branchial arches and grooves or clefts. (Slides)

Pathology. Lateral cysts and fistulae arise from persistent thymic or branchial strands which may retain their embryologic tendency to vacuolization and canalization. Cysts and fistulae may occur anywhere along their tracts, although the cysts are more commonly found in the upper third of the neck. Fistulae may be present at birth or they may break through as a result of infection in a cyst. They may be complete or incomplete. When incomplete they may open above internally in the posterior tonsillar fossa, while below they almost constantly open externally along the anterior border of the sternocleidomastoid muscle near the clavicle. When complete, a rather uncommon occurrence, the fistulous tract travels a fairly constant course. From its inner opening in the tonsillar fossa it passes under the angle of the jaw, thence downward and outward under the midportion of the posterior belly of the digastric muscle, passing below the facial and above the glossopharyngeal nerve to come into very intimate relationship with the sheath of the carotid artery and the jugular vein.

This constancy of relationship with the large cervical vessels was strongly stressed by Senn. Failure to keep this in mind has been the cause of panic and disaster in many operations for the removal of these cystic tumors or their vestigial relatives, the lateral fistulae.

The lowermost opening of the lateral fistula is probably determined by the constriction of the canal by the bony opening of the chest which favors the breaking through of the usually infected contents at this level. The walls of these fistulae are often much changed by infection. Histologic investigation of these fistulae and cysts has shown their similarity to the histologic elements of the thymus anlage. One commonly finds areas showing either stratified or squamous epithelium. (Slide) The walls may contain encysted muscle fibers, cartilage and mucous glands. Lymphoid tissue is commonly present in large amounts. Leucine, tyrosine, cholesterin, myelin, palmitin and stearin have been found in the aspirated contents of the cysts.

Symptoms. The clinical picture of these cystic tumors is that of a swelling on the side of the neck, usually causing no symptoms unless it reaches such a size as to press on other structures, when there may be hoarseness, coughing, dyspnea and dysphagia. Pain and tenderness are not common unless there is infection. Fistulae may be present at birth or a cyst may become a fistula when it ruptures spontaneously or is incised. Repeated secondary infections may take place, in which case there is chronic induration and purulent discharge. The cyst may vary in size with the periodic drainage, and in elasticity according to the amount of fluid it contains and the degree of pressure under which it is held. In most reported series fistulae have been twice as common as cysts.

Differential Diagnosis. The correct diagnosis of a lateral cyst is very important if one is contemplating operative removal of a tumor mass in the angle formed by the anterior border of the sternocleidomastoid muscle and the lower border of the inferior maxilla. The close proximity of these cysts to important structures in the neck makes it imperative that the operator be acquainted with the hazards accompanying their removal. In many instances a direct diagnosis is made by exclusion of all other forms of tumors.

1. *Carotid Body Tumors.* These are usually solid tumors which are constant in their location in the bifurcation of the common carotid artery. They are probably related to the carotid sinus. Seventy per cent develop after middle life (forty to seventy years). They are very slow of growth, as a rule, and are freely movable from side to side but fixed to the carotid so as to prevent movement in the line of vessels. They are painless and not attached to the skin. They may be compressed easily and a bruit and thrill as well as distinct pulsation may cause them to resemble an aneurysm.

2. *Tuberculous Lymph Glands.* It is seldom one meets a case in which extensive pathologic change is confined to the body of a single lymphatic gland. Confusion most often arises when a small group of fused glands has broken down and is semifluctuant. In tuberculous glands the skin early becomes dusky red and is commonly fixed to the tumor mass. Carp and Stout state that an x-ray examination should be made of suspected cysts and that if calcification is shown it is suggestive of a tuberculous gland.

3. *Cold abscesses* from burrowing of pus along the fascial planes of the neck, and usually originating in tuberculous disease of the cervical vertebrae, may present a lateral cystic swelling in the neck. Aspiration and animal inoculation may be done in making a differential diagnosis.

4. *Aneurysms.* The presence of a distinct transmitted pulsation from the carotid vessels may sometimes be confusing in lateral cysts and lead to a diagnosis of aneurysm. It must be borne in mind that the pulsations are never expansile in lateral cysts and bruits over them are quite uncommon. Pressure on the carotid artery below the suspected tumor does not affect its size unless it is an aneurysm. Careful aspiration with a very fine hypodermic needle is permissible even when one suspects an aneurysm. Branchiogenetic cysts are most common in children, in whom aneurysms are exceedingly rare.

5. *Dermoid Cysts.* These are commonly attached to the skin and on aspiration the contents show the presence of products of the secretion of the skin and organs which it contains, hair follicles, sweat and sebaceous glands. The wall of a dermoid cyst will show lanuginous hair and hair follicles, which are never present in branchiogenetic cysts.

6. *Angiomas.* These are common in children and are usually present from birth. They are easily compressible and rapidly refill on release of pressure. This usually serves to differentiate them from branchiogenetic cysts.

7. *Cystic Hygromas of the Neck.* These are commonly noted at birth as soft, elastic swellings which may be unilocular or multilocular. They may be of large size at birth or their increase may follow irritation, infection or intercurrent disease. Aspiration before infection usually reveals a thin serous fluid which may contain endothelial cells. Histologic examination of the cyst wall will reveal the endothelial character of its lining.

8. *Aberrant Thyroids.* Cysts arising from aberrant thyroid rests can be differentiated only by histologic examination.

9. *Cervical Node Metastasis.* (See Slide)

MEDIAN CYSTS AND FISTULAE

Median cysts and fistulae are more easily understood on the basis of our present knowledge than the lateral cystic tumors. They arise along the course of the descent of the thyroid anlage, which first appears as an unpaired median branchial epithelial body. Its original site remains marked by the foramen cecum in the tongue.

They are commonly referred to as thyroglossal duct cysts and fistulae. At no stage of development, however, does the thyroglossal tissue have a lumen. However this thyroid anlage may vacuolate and cause cysts. The tract is usually lined with squamous or ciliated epithelium. (Slide) These cysts may be found anywhere between the foramen cecum and the episternal notch, but the majority occur below the hyoid bone. In some cases failure of the descent of the precursor of the thyroglossal strand may have resulted in the development of thyroid tissue in the base of the tongue near the site of the usual foramen cecum. The removal of such tumors has led to myxedema in some instances, especially where the tissue removed seemed to represent the functionating portion, if not all, of the thyroid gland.

The cysts are slow growing as a rule and rarely reach the size attained by the lateral cysts. Due to their location on the anterior surface of the neck they are subject to constant irritation by clothing and collar bands and often become inflamed and infected. When these cysts break through the skin surface as a result of such irritation, infection or trauma, they present a smooth, shiny lining which discharges a glairy, mucoid secretion often mixed with inflammatory exudate. The median fistulae arising from the discharge of the cyst contents on the skin surface are never complete, that is, they can never be traced uninterruptedly to the foramen cecum. Those cysts in the midline above the hyoid bone may discharge on the dorsal surface of the tongue near its base. After the cysts discharge their contents, the site of discharge may remain as a fistula for a varying length of time; then the opening may close and gradual reaccumulation occur. Such repetitions of filling and emptying are quite characteristic of these cysts. They are most commonly confused with the following conditions:

1. *Sebaceous Cysts.* One may be misled by sebaceous cysts if they occur in the midline, but the character of their contents leaves little doubt of their nature. Histologic examination of the cyst wall will show their primary dermal origin.

2. *Ranula.* Simple sublingual cysts usually elevate the tongue and are translucent in appearance. They are commonly confined to the bursae in the loose tissue of the floor of the mouth. It is possible that some of the expansive types may be derived from a distention of the epithelial cysts which are carried from the base of the tongue with the descent of the thyroglossal strand.

PAROTID GLAND TUMORS

Most parotid gland tumors are the so-called mixed tumors,—they are benign in that they rarely metastasize, malignant in that local recurrence is a predominating characteristic.

The embryology and pathology of these tumors remain without a thoroughly satisfactory explanation, in spite of extensive study. Some investigators define them as adenomas, while others maintain that they cannot be considered as such because of peculiar stromal changes, and suggest that they are true mixed tumors developing from embryonic tissue.

The tumors were at first thought to be purely epithelial in origin. An endothelial theory was later set forth but has been abandoned and the epithelial origin is now generally accepted.

Some authors maintain that spontaneous malignant change in a mixed tumor is so rare that for practical purposes it may be ignored, while others have found that as high as 20 per cent of these tumors become malignant.

The natural course of these growths is an increase in size at varying rates, leading to a corresponding degree of deformity, but otherwise causing very little disability as a rule. Pain and tenderness are rare and when present it is usually in a recurrent tumor or a very large one. Facial paralysis may result from pressure on the facial nerve if the tumor attains a very large size.

The diagnosis is usually possible by physical examination and a history of long duration.

TREATMENT

It may be stated that practically all lateral and median cysts and fistulae may become troublesome, most often from infection, and cases are on record where they have been the seat of malignant growth. Nothing short of complete eradication of the walls of the cysts and complete extirpation of the epithelial tracts of the fistulae can be considered as adequate treatment.

Some authors have contended that because of the high rate of recurrence and the dangers of facial palsy attendant on complete eradication, parotid gland tumors should generally be left alone. There is, however, a growing feeling that recurrence has been due largely to surgery that has been too conservative and that more radical operative treatment will give a much better prognosis.

Let us consider the operative treatment of these cysts, fistulae and tumors in some detail.

Lateral Cysts. In these cysts one must be familiar with the difficulties of dissecting in the triangles of the neck and must be skillful in the management of injuries of the large vessels in this locality. It is especially important to know that these cysts are often so intimately attached to the internal jugular vein that in some instances they may require the sacrifice of a part of the vein wall or even the extirpation of a segment of the vein. (Slide) It must also be borne in mind that the aspiration of air following injury of the large veins of the neck may become a serious problem.

I believe it is the common practice of most operators to dissect the neck with the head and shoulders elevated above the general body level. This position, while advantageous to the operator, favors the development of air block in the cerebral circulation which may terminate fatally for the patient if the block is extensive or exists for any period of time. The elevated position of the head and neck lessens the congestion of the veins in the neck dissection, but greater safety will probably be had if the surgeon works with the patient's head slightly lower than the general body level. This position is more favorable because of the disposition which the circulation makes of air aspirated through an injury to one of the large veins. Instead of the aspirated air being carried to the important centers within the cranium, it finds its level in the extremities where little damage ocurs and where its absorption takes place uneventfully. A further advantage in the head-low position is found in the distention of the readily collapsible veins by gravity. They become more easily recognized and thereby less often accidentally injured. This head-low position likewise favors the drainage of mucus and pulmonary secretions.

Lateral Fistulae. It is important in these conditions to establish the tract of the fistula before operation, if this can be done. Many ingenious methods have been suggested for following these tracts. I have favored the injection of the tract with a solution of hydrogen peroxide to which a few drops of methylene blue have been added. This solution, when introduced with a blunt-pointed syringe at a very low pressure, on coming in contact with the secretions of the tract, favors the formation of free oxygen which will distend the tract. The methylene blue being carried along by the gas bubbles will stain the lumen a light blue color. The escape of the blue gas bubbles from the pharyngeal opening in the posterior tonsillar fossa can usually be easily seen.

This slide (Slide) shows the usual incision and the character of the dissection of the lower portion of the lateral fistulous tract. The greatest difficulty is encountered about the level of the greater cornu of the hyoid bone where the fistula passes beneath the posterior belly of the digastric muscle. Blunt dissection at this level is probably the safest procedure. One should exercise care at this point not to injure the hypoglossal, glossopharyngeal or facial nerves. After the tract has been traced beneath the posterior belly of the digastric muscle, it is well to cut off the lower portion of the tract, leaving a small segment of the proximal stump. Von Hacker suggests passing a probe through the pharyngeal orifice of the fistula at this stage and then along the tract to the distal end of the remaining short stump. A ligature then transfixes the stump and is attached to the probe which when withdrawn into the tonsillar fossa will invaginate the fistulous tract into the posterior tonsillar fossa, where it may be ligated close to its base and the remaining portion cut off. (Slides) Some operators suggest removal of the tonsil on the side of the fistula with a dissection of the fistulous tract up to its inner orifice, and finally encircling this orifice with complete freeing of the tract. The opening left is then closed with a suture or two. This, to my mind, adds considerable gravity to an already formidable procedure and should be done only in those cases where its execution is easy. Koenig has suggested a method of dealing with the proximal stump where it cannot be invaginated into the pharynx. He recommends drawing the distal end of the short stump into the anterior tonsillar fossa through an additional slit opening followed by the fixation of the fistulous tract with a suture. This procedure will bring both openings of the tract within the pharynx and will prevent discharge of the contents of the fistula externally since both openings of the epithelial tract will be located on the same side of the pharynx in the tonsillar fossa.

Median Fistulae and Cysts. These may be traced in the same manner suggested for lateral fistulae. If they are well stained with methylene blue, as suggested, their outline may be more easily followed. Most operators favor a transverse incision. While I have had very good results with a vertical incision extending for a short distance from either end of an encircling incision about the fistulous opening or cyst. The transverse incision gives a better cosmetic result. The walls of these median cysts and fistulae may be thickened from recurrent inflammations, but they are often so delicate that only very careful sharp dissection will accomplish their removal without tearing into the cyst or tract of the fistula. The continuity of the cyst wall or fistulous tract is quite easily lost in those which have not been thickened by infection. When the hyoid bone is reached in the dissection of the fistulous tract, it is best to avoid trying to follow the tract beyond this point before dividing the hyoid bone. I have found it much easier to trace the tract to the lower margin of the hyoid bone and then immediately divide the hyoid on either side of the attachment of the fistulous tract. (Slide) After this is done, the tract is pursued upward and backward toward the foramen cecum. My experience has left me in doubt about one's ability to trace the tract for any distance beyond the hyoid bone with any degree of certainty. I feel that from the hyoid bone onward one must rely on simply coring out the tract in the tongue muscles in the general direction of the foramen cecum. The direction and distance may be easily visualized by referring to the hemisection of the head and neck shown on the slide. (Slide) In most instances one had better avoid entering the oral cavity. Careful hemostasis should be secured and no drain inserted in these cases unless oozing is intractable. The fragments of the hyoid bone, which are left after the symphysis has been excised with the tract, may be approximated by a suture above and one below the bone. When these sutures are tied, the ends of the hyoid bone are brought into close proximity and fibrous union will usually result. After the first few days no difficulty is experienced by these patients due to the disposition of the hyoid bone. At first some slight pain may be experienced with swallowing movements.

Parotid Gland Tumors. Hamilton Bailey has pointed out that certain radical alterations must be made in our current teaching and practice with regard to the treatment of these tumors. The most important modifications he lists are: (1) Most tumors of the parotid are radioresistant. Deep x-ray therapy has proved futile if not harmful. (2) The capsule of encapsulated tumors must be removed. (3) Adequate exposure should be employed with an incision giving good access to the whole of the gland. It has been contended that the small rather than moderate-sized or large parotid tumors tend to recur, although other surgeons dispute this. With an inadequate exposure the smaller the tumor, the more difficult it is to be certain it has been removed properly. If the tumor proves non-encapsulated, incision should be prolonged downward and the external carotid artery ligated to attain comparative avascularity. (4) The surgical anatomy of the parotid gland needs revision. It consists essentially of a large, superficial and a small deep lobe; these are joined by a comparatively small isthmus which is variable in size but rarely absent. The main parts of the facial nerve—the temporofacial and the cervicofacial—embrace the isthmus, but no part of the facial nerve actually runs within the gland substance. The facial nerve should be looked upon as meat within a parotid sandwich, its main divisions lying between the deep and superficial lobes; but because of the small size of the deep lobe, the outlying subdivisions of the nerve rest between the superficial lobe and the masseter muscle. (5) Even when facial palsy occurs, the deformity can be mitigated by performing Lodge's operation, inserting a fascial strip subcutaneously. If surgical treatment is withheld because of the fear of facial palsy, the paralysis may ensue anyway from pressure of the growing tumor on the nerve.

I should like to conclude by showing you a few slides of patients I have operated on for these various tumors of the neck. (Slides)

DIAGNOSTIC SURGICAL CLINIC

Indications for Operation in Gastric Lesions

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I should like to preface my remarks by a few generalized statements. In general, the indications for surgical treatment of gastric lesions are really confined to two groups: (1) tumors, and (2) complications of peptic ulcer. The complications of peptic ulcer are somewhat dramatic, as you know. They might be listed as perforation, obstruction, hemorrhage, and intractability.

I will mention a few points about gastric ulcer a little later.

In perforation, immediate operation is, of course, strongly indicated. There will practically never be any doubt about that except from the standpoint of diagnosis. We must remember, however, that about twenty per cent of patients with perforation do not have a history of peptic ulcer, so therefore we do not consider a positive history as a prerequisite to making a diagnosis of perforation.

I want to call your attention particularly to an error commonly made in the interpretation of the collapse which occurs in perforation, as due to shock. This is not shock at all, although some physicians erroneously call it that. True enough, the patient may fall to the ground or the floor when his perforation occurs, but if you examine him you will find that his blood pressure is normal and his pulse rate is practically normal, perhaps ranging from 85 to 90, and obviously he is not in shock. Contrast that to the collapse which occurs in hemorrhage and you note a distinct difference; in fact, you can make a diagnosis on that one point alone. In case of a hemorrhage from a peptic ulcer you will discover that perhaps the patient or the relatives of the patient give you a history of the patient's having walked to the bath room after feeling a little weak or nauseated and after searching through the bath room cupboard for certain medicines, finally becomes so faint that he collapses to the floor; he faints, and about that time the rest of the household has arrived. When you take that patient's blood pressure it will be found down to 70 or 80, and the pulse rate around 120; he is in a true surgical shock.

I should like to call attention particularly to that distinct differentiation between the types of collapse in those two conditions.

I am unable to give you specific data regarding the indications for operation in hemorrhage. Opinions differ as to whether we should do immediate operation for an acute bleeding ulcer or whether we should treat that patient conservatively. I believe that Finsterer, a Vienna surgeon, first called our attention to the possibilities and advantages of operating on these acute cases. I do believe, however, that if we operate on them we must do that within the first forty-eight hours. In fact, I am convinced of that. You dare not procrastinate two or three days after a patient has had three or four hemorrhages and finally say, "Well, I guess we are not going to cure you by medical treatment so we will try surgery." That is fatal. That must not be done. If you operate upon that patient you must do it within the first forty-eight hours; you must have a series of eight or ten donors lined up in order to keep the patient out of shock on the operating table, and bring him back to a safe status postoperatively. Moreover, the surgeon must be a well-qualified surgeon, because most of these bleeding ulcers are penetrating posteriorly, and it is no easy trick to get in back behind the duodenum and get the responsible arteries, or to ligate the bleeding point in the ulcer. So surgical treatment of a bleeding ulcer requires expert surgical attention.

I should like to confine the rest of my time to our patients. This is patient No. 1, a man fifty years old who entered our hospital complaining of recurrent epigastric pain of twenty years' duration. Originally the pain was dull, gnawing and burning in character, but disappeared completely following alkalies or bland foods. He frequently awakened about two a. m. with pain, which would persist unless food or alkalies were taken. As you know, this is a fairly typical reaction in patients with peptic ulcer; they are awakened about two a. m. with their distress. There have been periods of intermission during which time the symptoms were relatively mild; there has been no nausea or vomiting until two weeks previous to his entry.

During the past several weeks previous to entry, the pain increased in character and had a definite tendency to be cramp-like. I want you to remember this because this really is a point which leads to a correct interpretation of his lesion. Alkali and food do not relieve the pain with the same degree of consistency as previously.

The vomiting which has occurred during the past two weeks, however, does relieve the pain. That of course is true of almost any type of vomiting. The patient feels better when he gets through with his vomiting.

He has had a weight loss of thirty pounds in four months and during this time he has tried many, many types of diet and has been under fairly good medical care. His son happens to be a physician and at least during the past few years he has had the value of his son's advice.

Physical examination was negative except for moderate epigastric tenderness. G.I. series showed a marked six-hour retention estimated at about 90 per cent, with failure to visualize the duodenal cap. The diagnosis was organic obstruction of the duodenum.

Additional examination revealed N.P.N. 43, blood chlorides 470, serum albumin 4.9, and serum globulin 2.4; red count practically normal, as were the other laboratory data, except for the gastric analysis, which revealed an increase in the acid. There was 40 degrees free acid and 24 combined, making a total acid of 64. With an alcohol meal the total acid rose to 128.

This entry which I am discussing was six months ago and we operated on him at that time, doing a typical posterior Polya resection of the stomach. He had a marked stenosis of the duodenal ring which would not admit a finger and in fact appeared to be almost complete.

In the history I want you to note the chronicity and the typical relief from alkalies and food, particularly during the first phases of the disease. That is typical, of course, of peptic ulcer with a diagnosis which is obvious. He has had fairly good dietary care without relief. When obstruction develops, alkali and food are not apt to afford relief. That, I think, is a very important point from the standpoint of the indication for operation. If you recall the history, I said that for the past month his pain had been much worse, cramp-like in character, such as that produced by intestinal obstruction. As you know, the pain of peptic ulcer itself is mild, but the pain of its complications may be extremely severe, perforation being worse and obstruction being less moderate.

It is true that the pain might be in the nature of a pylorospasm, but if it is, good management for a few days, including diet, sedation and rest, will give the patient relief. If you find obstruction on x-ray due to pylorospasm, a few days later with treatment you will find that the pylorus is again opened and he has no six-hour retention.

Another astonishing feature is the small amount of vomiting in this case, considering the fact that he had an obstruction which was practically complete.

For your sake I would like to ask him to describe the amount of vomiting he had. Did you vomit every day?

PATIENT: I don't think I did.

DR. COLE: Not that often? How much at a time? Half a cup?

PATIENT: Probably it was over a glass.

DR. COLE: That doesn't indicate very much retention, does it? A casual question like that might give you a misleading interpretation of his condition. You might say, "Well, he didn't have much obstruction." Let me warn you that that is not an indication of the degree of obstruction unless you go back and find out how much food he has been taking. I predict during the time he was vomiting you will discover he was taking only about a third of his required intake. Were you eating every meal?

PATIENT: I tried to but couldn't.

DR. COLE: They frequently say, "Yes, I ate every meal, Doctor," and only when you confront them again with more questions as to exactly how much they did eat do you discover that the man was automatically starving himself. Did you find that was true?

PATIENT: Yes, sir.

DR. COLE: That you would eat a few bites at one meal and call that enough because you couldn't eat any more?

PATIENT: That is right. There wasn't much desire, to be truthful.

DR. COLE: Regarding the indications for operation in obstruction, I suppose that some of the internists might say that they would like to take many of these patients and treat them conservatively for a time. I agree to that if there is a possibility of the obstruction being due to pylorospasm. As soon as I have proved that the obstruction is not due to pylorospasm I then am willing to recommend that the patient is a candidate for surgery, because, true enough, you may be able to treat him moderately successfully for several weeks or months, but at the end you are not going to improve that scar formation, and the patient is going to have a much worse degree of obstruction at the end of a few months than he had before you started; you are putting the surgeon behind the eight-ball in so far as the patient is probably a poorer risk then than he was when you first saw him.

I think the chance of medical cure in the presence of obstruction which is already definite is quite small. The mortality rate in that group of cases is so small that I think we have very little to lose by operation. The operation has certain advantages and of course certain disadvantages. There is obviously a definite mortality rate associated with the operation. In 30 or 35 resections performed for duodenal ulcer during the past five years, we have had a mortality rate of 6 per cent; so we have to keep that in mind as one of the disadvantages of operation. But of course there is likewise a mortality rate in the medical treatment because obstruction will keep on progressing sometimes in spite of good medical care.

Our patient under discussion had an attack of so-called indigestion a few weeks ago, and I would like to have him tell you about that. You had some attacks that made you think you were going to have your old trouble back again, is that right?

PATIENT: That is right.

DR. COLE: He came in to see us and was much disturbed, and after we heard his story, which was largely a matter of nausea and much burning—

PATIENT (Interposing): I didn't have much burning but you thought I should have another diagnosis.

DR. COLE: How are you feeling now?

PATIENT: Wonderful.

DR. COLE: We looked him over and he had no obvious explanations such as postoperative hernia, obstruction, etc. to cause his symptoms; accordingly I flatfootedly told him he was going to be all right, and that this was not a recurrence of the ulcer. How did I know he was not going to have a recurrence of his ulcer, i.e., a jejunal ulcer? This is my explanation of that. When we have a duodenal ulcer with a high acid, we do a very high resection, a resection so high that you can't do it outside the abdomen; I am convinced in my own mind that that patient is not going to have a jejunal ulcer. I realize that is a very dogmatic statement, but I personally have never seen a jejunal ulcer following gastric resection. I know they occur; they are reported in the literature, but I have never seen one of them, and I attribute that largely to the fact that we do such high resections that there is not enough stomach left to give rise to hyperacidity. I was perfectly sincere in telling him I did not believe he was going to get a recurrence of his ulcer, that it was a temporary upset due perhaps to worry or dietary indiscretions. However, he said he got over that disturbance very shortly and is now feeling wonderfully well.

I want to ask him again a few more points about the medical care which he needs now, compared to before operation. How about that?

PATIENT: I don't need any care at all.

DR. COLE: You don't need any at all?

PATIENT: May I say something?

DR. COLE: Yes.

PATIENT: In the last twenty years if I loaded up all the stuff that I have taken it would fill a 200-pound barrel.

DR. COLE: I think he is truthful in that.

PATIENT: After the operation I think about two weeks after I was home I had a little distressed stomach and I called and they said to take a quarter of a teaspoonful of baking soda, and I haven't taken a thing since. It is just six months since the operation.

DR. COLE: What do you eat?

PATIENT: I eat everything now but fried stuff and spicy stuff.

DR. COLE: And none of us should eat the spicy stuff anyway, is that right?

PATIENT: That is right.

DR. COLE: We told you about a few other things you shouldn't eat in the way of raw vegetables, particularly raw cabbage?

PATIENT: I don't touch it.

DR. COLE: The limitations on his diet are very trivial indeed. In fact, we tell him to eat anything that an average individual should eat. I am not sure that anybody should eat a large quantity of *raw* cabbage or similar bulky vegetables; there is not much food value in it, and not too many vitamins. Do you drink beer?

PATIENT: No. I have a question on that, though.

DR. COLE: All right, what is your question?

PATIENT: I never was a drinker, Doctor. I don't think I have drunk a half a pint of liquor in all my life, but I do like a glass of beer occasionally with a hot meal. I am not the kind who will get into the bar and have a beer because I am in with a bunch that like to drink. I did inquire at the hospital one time after I came back from my vacation. I said to your assistant, "I would like a nice glass of beer once in a while."

He said, "Only one?"

I said, "That's all."

He said, "Well, what else would you like?"

I said, "Well, I would like a cigar occasionally. I do smoke good ones."

He said, "How many cigars do you smoke?"

I said, "About three a day. I would cut it down to one."

He said "Well, you want one glass of beer and one cigar."

I said, "That is right," and he said, "Well, I think you can do without both of them." And I have lived up to it.

Dr. Cole: I think my assistant was being ultraconservative or more accurately was confining his remarks to your immediate convalescence. At any rate, I would say that now you could have a glass of beer and one cigar a day certainly. He can have beer, and smoke in moderation, providing we have done a high resection. So you go ahead and take your beer and one cigar a day.

Case No. 2 is a man sixty-six years of age. He entered the hospital with a complaint of epigastric pain of five weeks' duration. Contrast that with this first man. This pain was likewise present at night, occurring particularly about one a. m. Food or alkali would relieve the pain. Since the pain began he noticed pronounced fatigue, even though he got nine hours' sleep. About three weeks ago he began vomiting two or three times daily, particularly after meals. However, the vomiting has diminished during the past week before entry. Recently he noted that his stools were black. He has had a weight loss of six to eight pounds during this illness.

I think we ought to ask him more about that blood in the stool. How many times did you have that?

Patient: Just once.

Dr. Cole: Just one attack?

Patient: For a week straight I had it.

Dr. Cole: Of course he didn't have much opportunity to have several attacks because he has a history of only five weeks' duration.

His physical examination was negative except for his findings related to an ulcer. X-ray revealed an ulcer niche on the lesser curvature. Gastric analysis revealed no free acid and a total of 34 degrees. The red count was 2.82 on admission. Blood chemistry, however, was normal. After several transfusions his red count rose to 4.6. He was considered a gastric ulcer probably, but in view of his low acid it was thought that there was a danger of carcinoma and he was not submitted to much medical care, but was submitted to operation instead.

At operation, which is now nine months ago, a small indurated ulcer with a crater slightly over one-half inch in diameter was found on the lesser curvature at the midportion of the stomach; a typical posterior Polya resection was performed. Microscopical sections revealed the ulcer to be benign.

Reviewing this case, he has a history which is typical for a peptic ulcer, similar to the first one, but he has much more vomiting. We should next inquire as to the severity of his pain.

How bad was your pain?

Patient: It was bad.

Dr. Cole: Quite bad?

Patient: Yes.

Dr. Cole: His ulcer is in the stomach and I think it is quite agreed by all of us that the pain of gastric ulcer is distinctly more significant than the pain of duodenal ulcer. There was a constant pain or a pain that came in spasms?

Patient: It came in spasms. It would come even during the night while I was in bed.

Dr. Cole: We can not tell from that history alone whether or not he had any obstruction.

What about the indications for operation here? I mentioned the fact that he had a low acid which makes you suspect the possibility of a carcinoma, but he has more indications than that for an operative correction. The history of hemorrhage, which must have been quite severe to bring his red cell count down to 2.82, is a strong indication for operation. This would be a stronger indication if he had had more than one hemorrhage, but I think that any patient who has had one hemorrhage of sufficient severity to produce such marked anemia is probably going to have more hemorrhages. With an operation we can eliminate those hemorrhages, and we think that we can offer fairly good prospects of relief from symptoms as well as complications.

We must remember that about ten or fifteen per cent of carcinomas of the stomach have a normal gastric acidity. However, this patient's gastric acidity is consistent with carcinoma.

The size of the ulcer frequently is of help in distinguishing a benign from a malignant lesion. In this case it perhaps was a little smaller than the average malignant crater which we see, but we must remember that all carcinomas must start early as a small ulcer and if you see them early, as we did in this man, we would expect the crater to be small at a time when he had symptoms of only five weeks' duration.

But more important still, we must remember that carcinomas will show a definite improvement in symptoms from good medical treatment. That is almost universal. Eusterman two or three years ago called our attention to that fact very forcibly, and in fact that the x-ray findings will show a regression in the size of the ulcer; this regression might mislead us very easily to the belief that the ulcer is benign and is becoming cured, whereas in reality it is a carcinomatous ulcer and all we have done is relieve some of the inflammation around it which allows a little decrease in size.

In summarizing this case, how do we decide upon an operative procedure? As I said, the hemorrhage itself is a strong point in favor of an operation, but I go still further than that. If he had had no hemorrhage, I would still advise operation for this man, particularly since he is in the carcinoma age. I personally would not advise resection in a young man twenty-two or twenty-five years old with a gastric ulcer. I would say indeed treat that man medically; he does not have carcinoma. But when the patient is in the cancer age, such as this man is, there is too much danger of a carcinoma, particularly when the gastric acidity is low. We take too much of a chance for the advantages we gain.

We are not much worried about the mortality rate in this type of lesion. We have our best figures in resections for gastric ulcer. In the past six years at the Illinois Research Hospital we have done about thirty-five resections for gastric ulcer and we have not lost any of those; this is consistent with the experience of others, in that surgeons have the best figures in that group of patients, largely because we do not have the duodenal stump complicated by the ulcer. In gastric ulcer we can close the duodenal stump very easily and safely. Most of the dangers, and most of the deaths, in resections for duodenal ulcer are due to leakage of the duodenal stump. A great many of these complications from leakage of duodenal stumps arise from the attempt to excise the duodenal ulcer. I personally do not think it is necessary to remove the duodenal ulcer; if the ulcer cannot be excised without jeopardizing safety of closure of the duodenal stump, I leave a stump of the pylorus about an inch or an inch and a half long for closure; however, it is necessary to remove the acid-secreting mucosa of the stomach, which can be done in a minute or two or less. It is very loose and removing it leaves plenty of pyloric wall to invert to give you safe closure. This closure is of course not as safe as closure in a man with an ulcer of this type (i.e., gastric). His duodenum is normal. His lesion is in the midportion of the stomach and we expect to get a perfect closure of the duodenum. As I have said, we have had no deaths in resection for gastric ulcer, whereas we have had two deaths in resections for duodenal ulcer.

Again, let's ask him how he has been since operation. How do you feel?

PATIENT: Very good.

DR. COLE: Since operation?

PATIENT: I feel better than I did ten years ago.

DR. COLE: Are you able to eat?

PATIENT: I can eat good.

DR. COLE: What foods don't you eat?

PATIENT: I can eat everything now.

DR. COLE: Everything?

PATIENT: Everything.

DR. COLE: Do you eat a lot of spicy food?

PATIENT: Well, not too much.

DR. COLE: How about raw apples? Have you tried them?

PATIENT: No.

DR. COLE: Raw cabbage?

PATIENT: Very seldom.

DR. COLE: I think he has remembered our precaution not to eat raw cabbage and perhaps raw apples. We let them try raw apples and if they can eat them all right, but if they discover it gives them a little dyspepsia we say don't eat them. After all, perhaps all of us have a few idiosyncrasies against certain foods. Under such circumstances, what do we do? We simply leave those offending foods alone. If they amount to three or four types of food, absence of which will not create an unbalanced diet, then the patient is no worse off than the rest of us. In general, we have very few restrictions in the diet of our patients who have had gastrectomies. We tell them to eat sparingly of raw vegetables and spicy foods, but to eat them if they create no digestive disturbances. We can really name on the fingers of one hand the things he cannot eat. After operation we therefore do not treat him always as an ulcer patient, and therefore I want to try to impress upon you that the old adage "once an ulcer always an ulcer" is not quite true. True enough, for two or three weeks following operation we watch the diet and intake quite closely to be sure that the stoma is open and functioning properly. Occasionally we will be driven to a modified type of operation; in that case, we will want to put more limitations on the person's diet, activities, etc.

There are one or two other types of patients I want to discuss. Let's discuss another patient who, unfortunately, is not here. This man was seventy years old. Onset of symptoms was ten years ago when he noted mild epigastric pain of a burning, gnawing character which was relieved when he would take food. The symptoms persisted, but were not severe enough to make him go to a physician. He had intervals of several weeks or a few months when his symptoms would be relatively mild. During the past year his symptoms have been more persistent, without periods of freedom from symptoms. Likewise, during the past few months the elimination of pain with food and alkali has been less consistent. There has been no vomiting at any time.

Physical examination is negative. The G.I. series showed an ulcer crater 2 cm. in diameter on the lesser curvature at the pyloric area. Gastric acidity, 28 total, no free acid. Blood chemistry normal. Here again on the basis of the G.I. series we concluded this man should have a benign ulcer, but decided we had better operate on him since he was in the cancer age and had a low gastric acidity. He had an indurated ulcer about the size of a quarter on the lesser curvature; from its appearance we could not tell whether it was benign or malignant (that is, grossly), but the microscopic section revealed adenocarcinoma. In this patient also, we did an extensive gastric resection, since carcinoma and ulcer alike require high resection, although for a different purpose. I would like to emphasize again that we cannot tell from the gross appearance of a gastric ulcer whether or not it is benign or malignant. Naturally, large fungating masses are almost always carcinomas.

Note the similarity of the history in this patient to that in Case No. 2, except that here the duration of symptoms is longer. That would suggest, of course, a benign lesion. Presumably this patient had a benign ulcer for years and sometime or other during the past few months a carcinoma has developed in the ulcer.

I think this happens in about ten or fifteen per cent of our carcinomas; at least ten per cent of them are carcinomas developing on top of a gastric ulcer. That is one of the reasons I think it is dangerous to treat patients with gastric ulcer, when they are in the carcinoma age; that is a terrific responsibility, since the error of treating a malignant ulcer conservatively as a benign one for only a few months may change an operable lesion with a good prognosis to an inoperable, hopeless lesion.

I don't know any way of accurately determining whether or not a small gastric ulcer is benign or malignant.

I think if I have two minutes left I will tell you about a young physician twenty-eight years old whose symptoms date back two years ago when he developed epigastric pain of a dull gnawing character. A moderate amount of belching was associated with the pain. The pain appeared one-half hour to an hour after eating and persisted until food or alkali was taken. Occasionally food or alkali would increase the pain, which, however, would decrease later.

He was discharged from the Navy on account of his symptoms which were diagnosed duodenal ulcer, and entered private practice. The symptoms persisted in spite of meticulous care of the diet. He noted that his symptoms would be much worse during periods when he was making many night calls and obtaining insufficient rest. If he took several days off, his symptoms practically disappeared, but recurred when he returned to heavy work.

His physical examination was negative except for a slight tenderness in the epigastrium. Gastric analysis revealed no free acid and a combined acid of 14 on one occasion and a combined of 28 on another. The gallbladder series was normal. The rest of his examination was normal.

On the basis of his low acid, his internist taking care of him thought that we should investigate the possibilities of other disease causing his symptoms; I agree wholeheartedly to that philosophy, particularly since his gastric acidity was so low, even though we had x-ray evidence of a duodenal ulcer. The point I want to bring out in this young physician is this: that his ulcer is classed as intractable. He has had good therapy in the past year and he still has symptoms. What should we do with such a patient? Is he a case for operation? When I first saw him I told him I thought he was, but that he should be hospitalized for a complete examination. When the low acidity was found, I became doubtful as to whether or not he was a candidate for operation.

When operation is the therapy of choice in these intractable ulcers, we do high resections. In this case, the resection (if done) must be more extensive than any other type of resection because the danger of jejunal ulcer is greater than in any other type; if it were not for the fact that he has this low acid, I would advise him to have a gastric resection. Although we may at a later date change our attitude and recommend gastric resection, I do not believe the chance of a cure is as good here as in the average case.

DIAGNOSTIC SURGICAL CLINIC

Diagnosis of Disturbances of the Sympathetic Nervous System

By MAX M. PEET, M. D.
*Professor of Surgery, University of Michigan School of Medicine,
Ann Arbor, Michigan*

I may say it is a stimulus to have an audience of this size at this time in the morning; nothing gives a speaker more pleasure.

I am presenting this morning a more or less heterogeneous group of patients. For many years we have had certain types of conditions of which we had no conception of their etiology or of their treatment. They had no grouping. In fact, they did not belong in any particular category of medicine. Sometimes they were surgical, some were pediatric, some were medical. No one had paid a great deal of attention to them, largely, I think, because no known treatment had been very successful. Now we are beginning to realize that a good many of these conditions are actually diseases of the sympathetic nervous system.

I remember quite a few years ago a few of us were visiting with Dr. Cushing at one of our meetings, and he said, "You know, boys, I think that sometime we are going to have text books written on diseases of the sympathetic nervous system just as today we have books on diseases of the spinal cord and of the brain. I think there is going to be a time when diseases of the sympathetic nervous system are of equal importance," and he added pathetically, "I am too old to take this up. It is up to you young fellows to carry on."

Now that that generation is the old one, our younger men are carrying on still more, and I think the advances in the surgery of the sympathetic nervous system will probably be written.

With all rather sudden changes in concept of disease and its treatment we have had many, many false leads, but some of those false leads were of great importance although at the time their significance was not realized. There have been certain articles by certain men advocating sympathectomies for this and that and the other, but Royal and Hunter in Australia really started our present-day conceptions. I admit that their tenets were entirely wrong at the beginning. They thought that sympathectomies would prevent or cure spastic conditions in the limbs of these poor cerebral accident children, and at first it did seem to do some good. You remember Royal and Hunter came to this country and demonstrated their work because the men in this country had not been able to duplicate it, and when Royal and Hunter were here they weren't able to duplicate it either. They operated here in Chicago and many other large cities, but the outgrowth of that which in itself turned out to be of no value was of the greatest interest and stimulus and many chance observations made at that time have now borne fruit.

The first group of cases which I will discuss (and we have one patient somewhat illustrating the condition) actually came from those observations of Royal and Hunter; that is, that when they had divided the sympathetic nerves to an extremity, in their case in the hope of overcoming spasticity, they noticed that the skin instead of being cold and clammy, as it is in so many of those children, was warm and dry. That was a chance observation. It had nothing to do with their idea that spasticity would be overcome, but the fact that these children did have dry, warm extremities immediately suggested that that might be a cure for Raynaud's disease. We have never had any concept of Raynaud's. It is a disease predominantly of young women, but it does occur in men. Perhaps ninety per cent of the cases occur in young women and five per cent in men, but don't be misled when you see a case in a man and say, "Well, this can't be Raynaud's disease because he is a male." Some of the most striking cases I have seen have been in men. Almost always they get these vasomotor changes with the fingers getting white or bluish or changing from white to blue and sometimes almost to purple, under emotional strain; any upset, it may be pleasant or unpleasant, any mental turmoil will bring on these changes, and any exposure to cold. Some of these people say, "I never go out in the wintertime without heavy gloves and heavy socks and overshoes because my skin gets so cold so quickly." When they get the changes they also get pain and the pain may be quite excruciating. Others will notice if they put on very heavy clothing to keep warm that they will be quite comfortable. So the treatment for Raynaud's has been, one might say, warm clothing or change in climate, but after the disease has progressed even a warm room might not relieve the patient.

I have seen patients in the hottest August weather suffering severely from pains in their feet and in their hands due entirely to vascular spasm. At first it had only been under emotion or under exposure, but finally as the disease had progressed, even a warm room and without any mental upset would give them these attacks.

Sometimes the condition is striking. I remember one prisoner who had been sent to us from our state institution because they had noticed at times the fingers of his hands became almost black, strikingly so. They were afraid of gangrene. Gangrene actually results in some of these cases. He was a young fellow twenty-five or so, and I took him in to show to my class. The room was warm, he was under no emotional strain. They did not know that he was a prisoner. There was no garb upon him that designated his status in society and there was nothing for me to show the class except just the patient. Of course, that helps a little bit in demonstrating. Then I turned, purely accidentally, you might say, and asked him what particular job he had been in before this last one. I knew that he had been sent up for highway robbery armed, which is still done in some parts of our state. There was just an instant of hesitation on his part, hardly enough, I think, perhaps for the students to realize that he had hesitated; there was a mental turmoil, shall I say. Then he said, "Garage mechanic," and I held up his hand to the class and said, "The best lie detector that has ever been invented." His fingers were almost black. After we got out I said, "Now, what *had* you been doing?"

He said, "Oh, the same thing, but I had never got caught before."

After a sympathectomy he no longer responded to any emotional strain or to cold. There was no danger of gangrene coming on in his fingers, but I also removed any lie detector apparatus which he had.

That is a striking case, but it illustrates very graphically a rather common condition.

When Raynaud's disease was first described, Raynaud considered it a disease entirely of young women, he said of emotionally unstable, hysterical type, but it has been found in staid Swedes, in Norwegians, in individuals that you just can't imagine becoming upset. It occurs in all races; there is no choice, but there is a definite sex preponderance.

This woman whom I am presenting has Raynaud's disease both of the hands and of the feet. I just saw her for an instant before I came in. She said that she was very willing to come in and talk to you. How old were you when you first noticed difficulty with your hands?

PATIENT: Twenty-seven years old.

DR. PEET: That is a little older than the average. I have seen it in fifteen, sixteen and seventeen-year-old youngsters more often than the older ones. What did you notice first?

PATIENT: First I had an infection in my finger from the tip of a needle and my whole hand swelled up and after the infection went down I noticed my hands were blanching and getting all white and blue and numb; I couldn't pick up a needle. So I went to Dr. de Takats and he said, "You have to have an operation." I was operated on the first time and after I went there the first time I had to have all three operations for both hands and both feet. The feet proved successful right away. The right hand kept bothering me so he went over it and operated on it another time. It still wasn't right so he operated on me through the back and it seemed to help a while, and in another year or so I was operated on once more through the back and I have been pretty good since.

DR. PEET: You noticed this blanching. You said your fingers would get white and bluish and they would change rather quickly, would they?

PATIENT: They would last for hours; they would not come back.

DR. PEET: I mean it would come on suddenly, or was it very gradually?

PATIENT: It would come on gradually.

DR. PEET: In most people it comes on rather suddenly. What would bring yours on?

PATIENT: Change of weather.

DR. PEET: If you went outdoors when there was snow on the ground would your fingers get white?

PATIENT: They could almost get white right now too.

DR. PEET: What did you do, have to wear warm mittens or gloves and stockings?

PATIENT: No, I wouldn't wear wool stockings; I would hold my hands tight.

DR. PEET: Your feet didn't bother you as much as your hands?

PATIENT: Not so much, my hands the most.

DR. PEET: Would you notice this also if you got worried about something or excited about something?

PATIENT: No.

DR. PEET: You didn't have any trouble that way?

PATIENT: No.

DR. PEET: Did your hands and feet become painful?

PATIENT: The hands became painful, and painful through the shoulders a lot, and the feet were always cold and numb like. There was one time I could hardly walk.

Dr. Peet: They were so numb?

Patient: Yes.

Dr. Peet: Was that because you were out in the cold at the time?

Patient: I was out in the cold at the time.

Dr. Peet: That is a rather common history. They just find that they can't go outdoors in very cold weather. She said she didn't put on heavy stockings. Of course most women won't, no matter how uncomfortable they are. That was the reason, was it?

Patient: I don't know.

Dr. Peet: Many will wear a muff for their hands but they still have their big toe sticking out of the end of their shoe.

Since the operation your feet have been warm and comfortable?

Patient: Very well.

Dr. Peet: And the hands still blanch?

Patient: This one only.

Dr. Peet: Those up close can see that there are some color changes there. This one is especially blanched right now. If we had some cold water to put her hands in they would probably get white. Do you have to be careful in washing?

Patient: I always use warm water; I am sure not to use cold.

Dr. Peet: You wouldn't risk it?

Patient: No.

Dr. Peet: She says that the operation for her legs was highly successful. The operation which was done for the arms and hands has not been entirely successful. That is a condition which has been noted regularly. No one understood quite what the trouble was. We argued among ourselves: Were we doing a complete sympathectomy? Were we missing fibers? Anatomical dissections were always made trying to find out where these sympathetic fibers went. Was it that taking out the upper thoracic sympathetic ganglionated chain would we miss some of the fibers? It wasn't that at all. We were doing complete operations in these patients, but the reason that we were getting perfect results in the legs and imperfect results in the arms was because in the legs we were doing a pre-ganglionic operation. In other words, we were dividing the sympathetic fibers, the autonomic fibers, before they entered their respective ganglia. In the arms we were doing post-ganglionic operations, taking out the ganglia and dividing naturally the fibers beyond as well. That was beautifully worked out by Cannon at Harvard years ago, but we had not applied his physiological experiments. For example, if you take a common white rabbit, laboratory bred, he is not scared of you, and hold up his ear to photograph after a post-ganglionic sympathectomy, you have taken out the cervical chain, we will say, and those vessels are nicely dilated. We will pretend that this rabbit is quietly eating a carrot, he is not concerned, and as you watch him the vessels on the side that had the operation are dilated beautifully, the other is normal; but as soon as you take hold of the ear to hold it up to photograph those dilated vessels, that little insult (he is not scared of you but he doesn't want to be handled) pours a little more adrenalin into his system and just as quickly as that circulating adrenalin hits the vessels in the ear those vessels clamp right down and you just cannot get a photograph of the dilated vessels in the ear of a white rabbit after a post-ganglionic sympathectomy, no matter how tame that rabbit may be. You have insulted him a little bit and out pours the adrenalin, just as it does in all of us under emotional strain. In other words, those vessels are hypersensitive to circulating adrenalin and sympathin.

If you do a pre-ganglionic, if you cut the fibers before they enter the ganglion and leave the ganglion in place, then the vessels stay dilated and you can hold up the rabbit's ear, and photograph with trans-illuminated light without any difficulty because no longer are those vessels hypersensitive to circulating adrenalin and sympathin. Oh, there may be a little, but not enough to have any effect. That is the whole truck.

In the lumbar region we had been taking out lumbar ganglia, two, three and four. Actually the ganglia to the legs occurred lower down. We were simply interrupting the tract, the fibers that came down from the cortical, so the ganglia to the legs were in place and we had no hypersensitiveness to the vessels, but when we took out the upper thoracic ganglia we divided the post-ganglionic fibers and the blood vessels in those fingers were hypersensitive to adrenalin, to sympathin, and so forth. So the newest operation that is being done is to divide the anterior roots as they come out from the vertebra for the second and third thoracic nerves; cut those, leave the ganglia in place, divide the chain below the third ganglion, and bring it up through the muscle. That works beautifully, except that there is a tendency to regeneration, and various methods have been devised in an attempt to overcome it. None of them has been entirely successful. Today we can say that we can vastly improve Raynaud's in the hands, we can cure it in the legs, and in most cases I think we do cure it in the hands, but not in all; some of them have regeneration.

(Slide) This shows a Buerger's disease. I will show the difference between the two. This is a typical Buerger's which is so commonly confused with Raynaud's. Buerger's disease occurs almost entirely in men, not necessarily so, perhaps 95 per cent men, 5 per cent women, just the reverse of Raynaud's. You have a spotty change in color instead of a uniform one. Generally the big toe will be involved the worst. You have these areas all along of patchy discoloration, and when you examine this patient carefully you find that instead of having a normal pulsation there will be no pulsation in the posterior tibial and anterior and dorsal vessels. That indicates that we are dealing with an organic obstruction. Actually, of course, it is a thromboangiitis obliterans, something that is actually blocking the blood vessels, not a sympathetic disturbance.

There may be sympathetic disturbance in addition and at first we were rather enthusiastic about operating upon these patients, doing a sympathectomy. Then there was a reversal and we did not operate upon any because when we had had gangrene of the little toe or the big toe, especially if they had injured it, had cut the nail too short and started up a little infection, sympathectomy did not prevent the advance of the disease, it did not stop the pain of the disease. We had to do amputation just the same. But Raynaud's is a bilateral disease. So is Buerger's, but Raynaud's appears on both sides simultaneously; Buerger's has a long waiting period; it appears in the right leg first; it may not appear in the left leg for three or four years thereafter; always bilateral eventually but with a long period between. So when we had done bilateral sympathectomies for Buerger's disease, thrombo-angiitis obliterans, we didn't help the foot which was already badly involved, but there is a good deal of evidence that we did help the foot that was just beginning to have trouble. I do think that there are certain individuals who have had a unilateral amputation who otherwise might have had a bilateral. It is not a cure by any means, but it does increase the collateral circulation.

With Buerger's you have an inflammatory process, an actual thrombosis blocking, but in the collaterals you may have spasm, and it is that that you overcome by sympathectomy.

Actually at Ann Arbor we operate upon very few cases of Buerger's disease, but we do feel that in some cases at least operation has prevented serious consequences in the early stages, that is the beginning process in the good foot.

This is the typical color of a Raynaud's in the foot, uniform, gradually fading out of the color, instead of spots. It is hard to say where the change takes place. It is most pronounced in the distal phalanges, in the toes or fingers, but as it goes up it gradually fades, and in such a foot you find that the dorsalis pedis and the posterior tibial vessels are excellent, they are pulsating normally. This is a case of spasm of the blood vessels, not an organic case, and in Raynaud's disease operation does help, in the leg it certainly cures.

I have never seen a case of Raynaud's in the hands, even when we had performed a complete sympathectomy, that is, we had taken out the ganglia, go to gangrene. The patients sometimes had what this woman has, some blanching of the fingers, but she has no gangrenous tips, as so many of them have without operation, and eventually they may actually lose the first or second phalanges in each hand, which are just withered away, dried up, gangrenous. That can be prevented.

Will you tell us about your condition? (This is a male patient.) Have you had operation or not?

PATIENT: A week ago.

DR. PEET: That is getting around pretty fast.

PATIENT: Last Tuesday.

DR. PEET: What did they do?

PATIENT: I can't tell you the name of it but it is the operation for Buerger's disease to help the circulation.

DR. PEET: Abdominal approach to the lumbar chain. May we see your feet? How long ago did you begin to have trouble? When did you first notice it?

PATIENT: About four and a half years ago.

DR. PEET: You had pain at first?

PATIENT: Yes, I had pain in it for a certain time and finally swelling developed, in the left leg first, which I have lost since; I have an artificial limb.

DR. PEET: That was four years ago?

PATIENT: About four and a half years ago.

DR. PEET: When did it begin on this side?

PATIENT: This began about a year after that while I was still on a crutch. The leg was O. K. and then it began to act up the same, not quite the same as the other leg because the other one you might say I had a stroke in it, it was quick one night, and I had trouble thereafter until gangrene set in. This one came on gradually.

DR. PEET: He has some marked discoloration of the ankle which doesn't go with Buerger's but there is no discoloration of the toes now, perhaps a little more bluish than they should be. They are warm, dry. Did you used to sweat a good deal?

PATIENT: No, not unless I had woolen socks on, a little.

DR. PEET: Not excessively?

PATIENT: No, but they were cold, and the main point was I couldn't walk any distance; they hurt to beat the band the minute I took a step.

DR. PEET: Now it is quite comfortable?

PATIENT: It is considerably. Of course, I can't vouch for the success of this operation as yet because it is only one week.

DR. PEET: We can't vouch for the success of it for two or three years because Buerger's goes so slowly.

PATIENT: The difference in blood circulation was noticed right away. It feels warm and comfortable and it feels that way while standing.

DR. PEET: Has there been a change in color?

PATIENT: Yes, there is a little change in color, although my color in the foot wasn't so bad as it might have been in some other cases.

DR. PEET: The toes haven't been purple?

PATIENT: The toes were not purple.

DR. PEET: Not like the other foot had been?

PATIENT: No. Of course the other one finally got gangrene.

DR. PEET: I do believe that this man will probably have his right foot preserved because he has had a sympathectomy early.

We have a number of other conditions which we operate upon, diseases of the sympathetic nervous system.

(Slide) Hirschsprung's disease, megalocolon, enormous dilatation of the large bowel, it may be of the entire large bowel, it may be of the ascending or descending colon. This boy was unusual in that he was seventeen years of age before he came to operation. Most of them come very much earlier. Most of them die before they are two or three years of age if nothing is done. Some of these children never have a normal bowel movement. This boy never had had. He came to us with enormous dilatation of the abdomen. His chest was pushed way out from the elevation of the diaphragm.

(Slide) This was taken shortly after operation. Instead of convex he is now concave. He still has this very marked bulging of his chest, and he probably always will have.

(Slide) This is an x-ray not of that boy but of a baby with megacolon that we had operated upon. This was a barium enema before operation.

(Slide) This was six weeks later.

(Slide) This was six weeks after that, three months following operation.

Hirschsprung's disease can be relieved, I think, in every case by lumbar sympathectomy. There is no question about it. These people can be completely cured. No longer is it necessary to take out the colon, which had a mortality of about 50 per cent. That boy that I showed you is now having normal bowel movements every day. We have had many others that have had the same experience, instead of a bowel movement once in five or six weeks and perhaps not then.

We have extended the operation to many other conditions. We use it in scleroderma. It is not a cure but it does improve the condition.

We use it in hypertension. We have now operated about 1500 patients at Michigan for malignant and essential hypertension. It is not a cure there, but it has given better results than any other procedure yet brought out.

The Speaker: At this time I want to express the Board of Trustees' appreciation to the Chicago doctors who have made this meeting so wonderful all the way through. As you know, we started off the first morning without a hitch, thanks to all those Chicago men who got up early to come down here to help us.

"The Nominating Committee appointed by Dr. Frank H. Lahey, President of the Interstate Postgraduate Medical Association of North America, recommends as President-Elect, Dr. Donald C. Balfour, Director of the Mayo Foundation, Mayo Clinic, Rochester, Minnesota.

"(Signed) Dr. E. S. Sullivan, Chairman
Dr. G. V. I. Brown
Dr. J. M. Dodd
Dr. H. G. Langworthy
Dr. T. B. Throckmorton."

At this time I want to announce that the last speaker on the program, Dr. Frank S. Dolley, Associate Professor of Surgery, College of Medical Evangelists, Los Angeles, California, sends the following telegram:

> "Incapacitation my associate Dr. Jones renders it impossible for me to attend meeting. Deeply regret inability to do so. Other two members group already in service."

DIAGNOSTIC NEUROLOGICAL CLINIC

Diagnosis of Peripheral Nerve Injuries

By LEWIS J. POLLOCK, M. D.
*Professor of Nervous and Mental Diseases, Northwestern University Medical School,
Chicago, Illinois*

Due to the fact that of course peripheral nerve lesions in civil life are rather rare, we will have to conduct what might be called a clinic from some lantern slides.

When a peripheral nerve is injured and impulses cease to travel through the nerve, there is a loss of function. This loss of function entails that of motion, sensation, and certain so-called trophic, sensory and vasomotor changes that lead to certain conditions the exact character of which is not known, but among them may be found such things as muscle atrophy and a change in the irritability of the muscle so that it does not respond to electrical stimuli as does a normal muscle.

It is necessary to examine for the loss of motion with some method of precision, so that you can follow a case from time to time to determine whether it is improving, recovering, whether it is stationary, since when it is stationary it leads to some indications for operative treatment. Therefore, some simple dynamometer is useful such as we illustrate here, an ordinary fish scale, by which the number of grams or pounds of pull might be registered on a graph, and this can be used for practically every movement in the body.

(Slide) The reason that it is necessary carefully to study motor movements is because of what are known as supplementary motions or trick movements, supplementary motility. For example, here we have a man who has a radial nerve palsy and you will note the wrist-drop which is characteristic.

(Slide) On this occasion we see that he has dorsiflexed his hand upon his wrist and the question arises: Shall we operate on this radial nerve or not? Is this a sign of recovery? This is not a sign of recovery. What he does in this instance is to close his fingers, to flex his fingers strongly, and if there is a slight shortening in the extensors of the wrist, then there is a passive extension at the wrist and that is a supplementary movement, and each nerve supplies muscles to which supplementary movements are added by the other uninjured muscles and it is necessary to know what these movements are so that you are not mistaken in a diagnosis.

What is true of motion is also true of sensation. We must have some standard methods of examination, and here is a method for the examination of pain so that each time you prick the man it is always done with a pressure that is constant. It may be simply built in that way, a winding of wire and an ordinary teasing needle sliding through a piece of glass tubing. In that way you will be able to outline the borders of sensory loss and determine whether or not a nerve is paralyzed completely or not.

(Slide) When you do that there is a characteristic appearance on the sensory chart some days after injury. When a man is injured, immediately afterward you will find there is a loss of sensibility to all modalities, to touch, pain, temperature, over the entire supply of the nerve. For example, if he had a median nerve lesion, that entire area would be anesthetic to all modalities. Some forty days afterward we find a shrinkage in the area of the loss of deep pin-prick and very hot and very cold stimuli. This occurs in characteristic zones always adjacent to uninjured nerves and is due to the overlap of function from uninjured and adjacent nerves. In some instances it assumes very large proportions. For example, here is an ulnar-median nerve lesion and this whole area has become innervated from the adjacent uninjured radial nerve. The appearance of a sensory chart of nerve overlap is very characteristic. There is always a zone in which there is a complete loss of all sensations, and then an intermediate zone in which deep pin-prick and very hot and very cold stimuli are felt. That is entirely different from the appearance of the sensory chart of a recovering or partial lesion which we will see later on.

(Slide) A very good method of quickly recording the appearance of a hand is by making a hand print and sometimes in the exigencies of war we are not able to take photographs of patients that will give us a very good record of the condition of the muscle. For example, here we see a very marked atrophy of the hypothenar eminence and we see a clawing of practically all the fingers, an atrophy of the thenar eminence. Here is another, a median nerve lesion, and we see the clawing of the middle and index fingers and a marked atrophy of the thenar eminence. There is a typical ulnar lesion with an atrophy of the hypothenar and clawing of the middle or ring and little fingers.

It is very difficult from the observation of the loss of motion and sensation to determine whether a nerve is sectioned or physiologically interrupted by compression or concussion. Some assistance is given to us by electrical examination and in addition to that which is known to you of the reaction of degeneration where the muscle fails to respond to faradic current because of the short duration of each stimulus since the time necessary for the stimulus to persist becomes lengthened more and more in degenerated muscles and that the muscle reacts sluggishly to galvanic current with a polar inversion, more critical evidence can be obtained by the use of the minimal current necessary to produce a contraction by galvanic stimulus. There it is found that when a muscle is completely denervated, has no supply at all from the nerve, the amount of current necessary is very little, as in this case of an animal that is completely denervated; up to 120 days you see that the amount of current necessary to stimulate is very small. The moment that recovery occurs there is a marked increase in the milliamperage necessary to produce stimulus.

(Slide) Another method for determining what nerve is completely sectioned or recovering is by using what is known as a progressive current. For example, a sinusoidal current, an alternating current, is a progressive current since it keeps increasing in time. When we do that very slowly, here is one in which the peak of the current took 3.8 seconds to reach, and when that is done in a completely denervated muscle we find that it takes a very small amount of current to produce a contraction.

(Slide) In contrast to that, this being the denervated muscle, that being the normal muscle, we see that when recovery occurs, the amount of current necessary at progressive currents of 1.2 seconds, at 2.8 seconds, at 3.6 seconds, at 8.8 seconds, becomes very large, 6 milliamperes as compared to a very small fraction of a milliampere when it is denervated. That is a very valuable method of making a diagnosis.

(Slide) The trophic changes that occur are those of the skin, and here we see a very marked keratosis and wrinkling, hardening of the skin. We see trophic ulcers. They all occur in analgesic zones and are due to some preceding injury: in ulnars, to flicking a cigarette ash, in median nerve, holding a lighted match and not noting that it is burning. The ulcer heals very slowly.

Nail changes also occur. Here we see some nail changes, new nails growing down and ridging.

Each nerve when injured produces a characteristic picture. This is a case of an ulnar nerve in which we see two things. We see an atrophy of the hypothenar eminence and clawing of the little finger, sometimes also of the ring finger, and an atrophy in the adductor of the thumb. On the reverse side we will see atrophy in the interossei muscles and particularly the first dorsal interossei, and clawing.

(Slide) This slide brings out the paralysis of the adductor of the thumb, which is supplied by the ulnar nerve. He is attempting to hold a piece of paper between the thumb and index finger, and we note how he flexes the distal phalanx of the thumb trying to use the long flexor of the thumb as an adductor. On the normal side the thumb is held extended.

Some supplementary movements occur, and here he is attempting to hold a book between the thumb and the index finger by means of the extensor pollicis. You see the atrophy in the interossei and the clawing in the ring and little fingers.

Remember what the appearance was of the nerve overlap in which there was a black zone like that and an intermediate zone, and compare it to what occurs when recovery is present. Here we see a recovery of touch over the isolated supply of the nerve. Touch never returns before recovery is present. Pain, however, does, and here we see pain has recovered in the isolated area of the ulnar supply which is the distal phalanges.

A very good sign of complete recovery is the ability to scratch the table with the little finger with the palm flat on the table, such as this man is doing; he is unable to do it on this side because he still has his ulnar nerve palsy.

Here we see a typical radial nerve palsy with wrist-drop, drop of the proximal phalanges of the fingers and thumb and the thumb is underneath the index finger because he cannot abduct the thumb in the plane of the palm which is due to the paralysis of the long abductor supplied by the radial nerve. Sometimes limitation of motion of other muscles than those that are supplied by an injured nerve occurs. This man, for example, cannot make a fist. Sometimes one makes a mistake in the diagnosis, believing that there is in addition to the radial nerve palsy also a paralysis of the ulnar and median nerves. That is not true, and none of us can completely close our hands into a fist with the wrist in a position of flexion. Here is an example of supplementary movement. Here is a wrist-drop. The man has extended his wrist by means of the pronator radii teres. We can demonstrate that on ourselves. If we flex our wrist and pronate the hand strongly we will find that passively the hand has become extended and one must be very careful not to interpret that as a sign of recovery.

When the lesion is high up in the arm, then of course we will have a paralysis of the brachioradialis and we can demonstrate that by having the man pull against some firm object on this side; we see the contraction of the brachial radialis and on that side none.

(Slide) This is the typical appearance of the overlap in a case of a radial nerve palsy, and that is the appearance in a recovering nerve. We see here the pain has recovered throughout. We have interlacing of the borders of loss of cold. Here is a recovery in a cleft. There is a marked interlacing of the borders of sensory loss, which is characteristic, seen here very well by the loss of cold in that area and the loss of touch over the whole area of the radial nerve.

A good sign of complete motor recovery of the radial nerve is to have the man stand, extend his forearm, supinate the hand, extend the fingers, the wrist and the thumb as in placing the inner side of his hand by the seam of his trousers.

In median nerve lesions the appearance of the hand is also characteristic in that the thumb is in the plane of the palm. Normally the thumb is held at right angles to the palm. Here, because of the paralysis of the short abductor of the thumb and opponens of the thumb, the thumb is in the plane of the palm. It is a simian, an ape hand. We also note the very marked atrophy in the thenar eminence, all of the muscles there being supplied by the median with the exception of the adductor of the thumb. Here is the typical appearance of the ulcerations that occur in a median nerve lesion.

That man cannot tense his palmaris longus, or produce that flexion of the index and middle fingers, and he cannot oppose the thumb to the little finger nor abduct the thumb at right angles to the palm. He cannot grasp a glass of water.

Another showing the characteristic appearance, the ape hand, the atrophy of the thenar eminence, marked keratosis, a healed ulcer. This shows his inability to touch the little finger with the thumb making the letter "O". In other words, he attempts to touch the little finger much as you do in a Boy Scout salute. That is a supplementary movement; marked atrophy in the thenar eminence and a straight thumb since he has a paralysis of the long flexor of the thumb. There is an ulcer.

Here is a characteristic sign. You can pick out median nerve palsies in large groups of women and men simply by having them make a fist. If they attempt to do so they are unable to flex the index finger and cannot flex the distal phalanx of the thumb.

Have them clasp their hands. The same symptoms appear. He cannot flex the index finger and he cannot flex the distal phalanx of the thumb.

The overlap sensory chart is very much like that that you see. Here is a median nerve, complete loss of sensation in a certain zone, an intermediate zone in which deep pain and very hot and very cold stimuli are felt.

When recovery occurs an entirely different picture ensues. Here is recovery in clefts. Here is a recovery of pain in the isolated supply of the nerve, a recovery of an island between two areas that are still anesthetic. There is an island of return of touch in the supply of the nerve. We know that he had a loss of touch here because he still has a loss of cold, so we know that touch has recovered in that area and that is typical of a recovering nerve. Here is an interlacing of the borders of the sensory loss of cold and of touch.

(Slide) Here is a completely recovered median nerve. He makes a fist; he completely flexes his index finger, and he flexes the distal phalanx of the thumb.

When both the ulnar and the median nerve are affected, then of course you again have an ape hand, the thumb is in the plane of the palm; it is quite flat, and the patient is unable to perform any flexion at all with the exception of slight flexion at the wrist by means of the extensor ossis metacarpi pollicis which is supplied by the radial nerve. That is a supplementary movement. All other movements disappear. This slide looks as if this were a plaster cast. That is because the appearance of this skin was that of a very thin parchment-like, pinkish, glazed thin skin, and that is known as glossy skin. It is the condition known also as causalgia. It was described first by S. Weir Mitchell, renowned neurologist in the Civil War, and very shortly afterwards by a Danish neurologist. It gives rise to the most excruciating pain I have ever observed in a patient. These patients are so affected that with this pain they sit in bed huddled with a protective attitude of the hands to prevent anyone from touching them; the least jar, a man slamming a door, a patient entering into the ward, any emotional disturbance, any sudden noise or a startle will bring on a paroxysm of such severe burning pain that it is unbearable. In the Civil War many of the patients who developed this in the lower extremities would urinate into their boots because they found by experience that moistening the extremity would diminish the pain. These soldiers will always have a wet towel about their hands or their legs so that the hand becomes often quite macerated.

You see some maceration there. The degree of motor injury is often so little that some of them have been wrongly diagnosed as hysteria. This is a very terrible and as yet unsolved part of peripheral nerve lesions.

Another ulnar and median nerve lesion (slide), not quite so severe because he can flex the proximal phalanges of the finger. Here is the typical overlap appearance of ulnar and median nerve lesion, and here is the appearance of a recovering lesion. Here is the appearance of a recovering lesion. Never do you have any return of sensation in the area between the supply of the median nerve and the ulnar nerve unless recovery occurs.

Here is a man with a musculor cutaneous paralysis. That is rather rare. Here we see on the right side that he is flexing his forearm and producing a contraction in the biceps. On this side you see how well the brachioradialis is supplanting the paralyzed biceps in producing flexion, and since the muscle is not bound down by the tendon of the biceps it is very broad when it contracts.

Sensory loss is about like that. Again you see there has been a shrinkage. That is an overlap.

(Slide) There is a deltoid muscle palsy due to a circumflex lesion, an axillary nerve lesion. He is unable to abduct his arm; he has paralysis of the deltoid and there is the sensory loss corresponding pretty much to the distribution of the fifth cervical segment.

(Slide) Here is a man of whom we could ask the question: Does he have a circumflex nerve palsy? I think our inclination would be to say no; he fully and adequately abducts the arm to a right angle, that is the function of the deltoid. This man does have a complete section of the axillary nerve and he is able to abduct his arm by supplementary movement.

In warfare, brachial plexus lesions do not resemble those in civil practice because of the type of injury, that is, they are gunshot and shrapnel injuries for the most part, whereas in civil life we see probably most of them as the result of childbirth and therefore we do not deal with such specific entities as Erb's brachial plexus lesions, upper brachial plexus lesions, or the Klumpke-Dejerine type, or the posterior cord type, although the residuals of the branchial plexus sometimes leave this picture. The best way of making localizations in brachial plexus lesions is to use a diagram to note the muscles that are paralyzed or weak, follow them up and for the assistance of the surgeon make a mark as to the place where the lesion will be found.

There is a brachial plexus lesion, and it affects chiefly the small hand muscles, and that would be one of the inner cord, Klumpke-Dejerine paralyses. Another looks very much like an ulnar median palsy. That is a lower branchial plexus lesion. Here we have the sensory loss that appears. This is the fifth and sixth cervical.

Here we have included also the seventh cervical and partly the sixth cervical.

There we have a man who has everything affected except the fifth and sixth cervical.

(Slide) In the lower extremities, often the femoral nerve is affected and gives rise to a characteristic gait; they either walk backward or always hold onto the thigh with the hand to keep the leg from flexing at the knee and giving way.

(Slide) Here is a man with a peroneal nerve palsy, external popliteal. He has a foot-drop; he is unable to dorsiflex the foot; he is unable to evert it. He can invert it by the tibialis posticus; that is supplied by the tibial nerve. This is his sensory loss.

Another one showing the sensory loss on the inner side. Here is another showing wide overlap. This is the only area in which he has complete loss of sensation. Originally he had a loss of sensation of touch clear up to there.

This is the ordinary overlap, the sensory chart, and that is the recovering nerve. There we see again the characteristic interlacing of the borders of the sensory loss. We see islands of complete recovery. We see islands of recovery to deep pin-prick.

Sciatic nerve lesion, a combination of the tibial and the peroneal. We have a complete loss of motion of the foot. Sometimes if the lesion is high the hamstrings are involved, although that is quite rare. Here we see in addition the man having the lesion high up into the buttocks, that the small sciatic nerve is also involved. It is purely sensory and gives rise to loss of sensation on the back of the thigh.

This is the inner side of the foot and shows loss of sensation.

Here we see something which you now recognize in my description of the painful lesion. You see the macerated appearance of the foot. That is a causalgia. Causalgia occurs in lesions of two nerves, the median in the upper extremity, the tibial in the lower.

Here is a characteristic ulceration that appears in sciatic nerve lesions. He probably injured the heel and afterwards it didn't heal.

This is the characteristic overlap appearance of the sciatic nerve lesion and this in contrast is the recovery, again interlacing of the borders of sensory loss, recoveries in islands, quite easily differentiated.

DIAGNOSTIC MEDICAL CLINIC

Congenital Hemolytic Icterus

By RUSSELL L. HADEN, M. D.
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This is a diagnostic clinic without a patient. I am sorry I cannot show you a patient today, but there is not much to demonstrate in congenital hemolytic icterus. I perhaps can tell you about it just as well.

Congenital hemolytic icterus, as the name indicates, is an inborn error; it is an anatomical error in which the red blood cells are spherocytes instead of normal biconcave disks. I can show you this abnormality of shape in bas-relief photographs, which illustrate an absence of the normal biconcavity of the erythrocyte.

As the name indicates, the patients are practically always icteric. If a patient comes to you, either a child or an adult, and tells you, "I have had jaundice from childhood or from birth, my brother or my sister has it, my father or my mother had it, my child has it," the first thing to consider is the possibility of congenital hemolytic icterus. The patient is seldom very sick. The time that the disease becomes active is very variable. We have done one splenectomy as early as three months of age, and numerous splenectomies as late as fifty years, so the disease may become manifest and require treatment at any time.

Typically, the spleen is enlarged also. A patient may present an enlarged spleen and have no icterus, because the icterus is variable. Both the size of the spleen and the degree of anemia are very variable. There are only those three characteristic features of the disease from a clinical standpoint: anemia, which is apt to be very chronic and to persist over a long period; icterus of varying degree; and an enlarged spleen.

This is a fairly common disease, much more common than is appreciated. With a typical family history in brothers and sisters or in parents, one should always consider the possibility of congenital hemolytic icterus. However, there are many cases of typical congenital hemolytic icterus in which there is no family history. This is true of other diseases of the blood, such as hemophilia. We not infrequently see patients in whom the correct diagnosis has not been suspected, but who have this typical clinical picture of chronic jaundice, enlarged spleen, and anemia.

The first slide is a normal blood film with the characteristic biconcave disks shown beautifully. The photograph was made by combining negative and positive plates.

The next slide is a blood film from a patient with congenital hemolytic icterus. Notice two things: The diameter of the cells is very much less than in the preceding slide. The two photographs were taken at the same magnification. Notice also that there is no biconcavity of the cells. This film is from a patient who long ago had her spleen removed for congenital hemolytic icterus. This anatomical peculiarity of the blood persists through the life of the patient, although there are no symptoms after splenectomy.

There has been much discussion about the cause of the disease. Some have thought that the unusual shape of the cell, the anemia, and other findings are due to a toxic process arising in the spleen or elsewhere. I am very sure that this idea is not correct, because we have now studied thirty-six patients after splenectomy, some of them for as long as fifteen years, and all have persistence of this peculiar shape of the cell. The cases who do not have this I am sure belong to an entirely different type of hemolytic jaundice.

The third slide I am showing to illustrate by contrast what the cells look like in ordinary hypochromic anemia. This is hypochromic anemia due to bleeding. Notice the wedding-ring shape of the cells. These cells are small and their diameter is decreased, but the picture as you see it under the microscope is entirely different.

There are a few other interesting things to be found on studying the blood in congenital hemolytic icterus. The patient has anemia, but the bone marrow is also overactive in an attempt to make up for the increased destruction of the cells. There is always an increase in the reticulocytes because of the hyperactivity of the marrow. The anemia is due to the fact that these spherocytes on entering the spleen are filtered out by the spleen from the blood stream. As a result of this, overwork occurs, and the spleen becomes enlarged. The jaundice is explained by the rapid and excessive destruction of the red blood cells. The bile pigment is formed more rapidly than the liver can excrete it.

The anemia may be due to the lack of balance between blood regeneration and blood destruction, although a mild case of spherocytosis does not necessarily show an anemia. At times also the degree of anemia may be very mild.

The family history of a patient with congenital hemolytic icterus is interesting. We have one family in our series in which I have personally studied the blood in four generations. So far as I can find, no one has previously ever actually seen and studied the blood of patients with the disease in four generations, although there is often a history for several generations.

Another interesting feature about this particular family is the fact that the fourth generation is the child of a mother who had her spleen removed ten years ago when she was ten years of age. When she got married and the family began to come, I watched with a great deal of interest to see if the disease would appear in the fourth generation. The first child was perfectly normal; the second child was brought in recently with a hemoglobin of 45 per cent and a marked spherocytosis such as the mother has. The film you have just seen on the second slide was from this mother a good many years after the spleen had been removed. So the fact that the spleen has been removed and the symptoms of the disease are relieved is no reason why it cannot be transmitted as well as if the disease has not been treated.

We have studied the blood in thirty-six patients who have had the spleen removed. In every case this peculiar characteristic of the cell, the spherocytosis, has persisted. It may not be exactly the same as before splenectomy, because anemia modifies the condition of the blood. The reticulocyte count returns to normal, and the icterus disappears entirely because the blood destruction ceases on removal of the spleen.

What I should like to emphasize is the necessity for keeping in mind the possibility of this disease. I am sure that many more cases will be recognized.

This next slide is another contrast I would like to emphasize. A normal blood is on my left as I face the screen, and in the center the cells of congenital hemolytic icterus are deeply stained because they are very thick; they are small cells both in diameter and in volume. On my right is another type of hemolytic anemia due to the excessive hemolysis of red cells after the inadvisable use of phenylhydrazine. Here the cells are entirely different.

The diagnosis of congenital hemolytic icterus can usually be made easily from laboratory studies. If it is an outspoken case, a trained laboratory technician can usually recognize the abnormality by simply looking at a stained film. In all cases we measure the diameter of the red cells by a very simple refractometric method. There are several types of apparatus on the market by which the measurement can be readily determined.

I have not mentioned the fact that these patients also show an increased fragility of the red cells; because of abnormal shape the cells are increasingly fragile in hypotonic salt solutions. They do not show increased fragility to other types of hemolysis.

The treatment of this disease is very simple—only splenectomy is of value. Nothing else does any good whatsoever so far as I can see; liver, iron, and other measures do not help a bit. Splenectomy cures the symptoms of the disease if the diagnosis is correct, although the spherocytosis persists.

When should splenectomy be done: We think a splenectomy should be done fairly early in life if the child has anemia or other symptoms of an active disease. Few children develop normally without a splenectomy; they are always anemic, they are always subject to infections, they never develop normally.

Dr. Tom Jones did a splenectomy on one of our patients, a child of four months who weighed seven pounds. She made a complete recovery. As a matter of fact, even infants with this disease get along remarkably well with splenectomy. We have never seen any difficulty with operation. Patients never have the complications that occur after splenectomy for other causes, particularly for Banti's disease.

I know of no disease whose course is more satisfactory than congenital hemolytic icterus after splenectomy.

DIAGNOSTIC UROLOGICAL CLINIC

Bladder Diseases and Their Treatment

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Diseases of the bladder in one short session is a big order, so I am going to touch the highlights and the most important diseases.

Let us take up first the subject of cystitis. Cystitis is a general term which covers many disorders of the bladder. The classic symptoms are practically all the same, namely: frequency of urination, painful urination, urgency, tenesmus, retention of urine or incontinence. The findings are gross hematuria, microscopic blood, bacteria, or pus in the urine. In addition to this sometimes there is partial or complete retention, and then supplementing it there may be incontinence of urine in the way of an overflow incontinence or an actual relaxation of the sphincter so that there is no urine retained in the bladder.

The diagnosis of cystitis rests upon the story which the patient tells and one must remember that the female is affected ten times oftener than the male, so that there are ten female patients with cystitis for each male patient that presents himself.

The value of a stained sediment of the urine to determine the invading organism is of great importance, and is done by cleansing the external parts, passing a catheter in an aseptic manner and obtaining a catheter specimen of urine which is centrifuged down and a smear made from the sediment. The specimen is stained and examined with the microscope, and when this is done, in about 75 per cent of the cases the infecting organism will be found. Very often the staphylococcus is at the bottom of the trouble, even though some colon bacilli are also found.

The treatment of the disease begins at that point when you have determined the infecting organism. We have found that the following simple procedure has stood us in good stead through the many years. If the patient after having this line of treatment does not clear up, in fact, if the symptoms do not completely disappear in three treatments, it is then that we should go further and begin to make a complete urological study to determine the source and origin of the infection.

This is the way the treatment is carried out. A soft rubber catheter is passed in an aseptic manner and the bladder is emptied. A 2 per cent solution of metycaine or a 1:1000 solution of nupercaine is instilled into the bladder in the amount of 10 cc. This is left in the bladder for five to eight minutes. The solution is allowed to run out, and the bladder is again empty. Next 10 cc. of a 10 per cent neosilvol solution to which has been added 5 to 8 drops of a 2 per cent silver nitrate is instilled into the bladder and allowed to remain there, the patient holding it as long as she can. After that, one of the sulfonamides (one of our favorites is sulamyd made by Schering and another is sulfathiazole) is given for a four-day period, one 7½ grain tablet three times a day.

Tuberculous cystitis is a type of cystitis that is usually very intractable. Commonly the patients come with a long history of having had cystitis which does not respond to any ordinary treatment. One should study these cases to find out if the acid-fast bacilli can be found, for then the diagnosis is established. It is necessary sometimes to treat a patient who has had a nephrectomy for tuberculosis of the kidney, and that patient may become a great care if he is not relieved of his terrible bladder disturbance.

We have found the following treatment of value: metaphen in Oil A 100 cc., anesthesin 1 gram and phenol 1 gram made up as a mixture and instilled into the bladder in amounts of 10 cc. allays the spasm and the discomfort of the patient with a tuberculous cystitis.

HUNNER ULCER

Another type of disorder which is very prevalent and frequently overlooked even by urologists is Hunner's ulcer. Hunner described the ulcer in 1910 and pathologists have never agreed that this is a true ulcer. It is characterized by pain which the patient can localize on the abdomen with the finger. You may ask the patient to localize the pain and he will point the finger to the exact spot, sometimes above the symphysis, sometimes down on the labia, but if you look at the interior of the bladder and localize the ulcer with a cystoscope you see that the area which the patient points to corresponds to that in the bladder.

The characteristic part of Hunner's ulcer is that usually the urine is clear, containing no blood cells or pus cells, and this is very deceptive because many physicians believe there could be no bladder disease unless pus cells or blood cells were present in the urine. However, these patients suffer from the ulcer even though there may be no sign of pathology in the urine. When the bladder is full, the most painful stage of this disease is reached. Most of these patients have to arise three or four times at night to urinate, and that is quite unusual in women, for if you are dealing with merely a hysterical type of disorder where the patient feels she has to urinate often, she will not have to arise at night for, as a rule, she will sleep soundly and not have to get up, so that this becomes one of the differentiating points. Of course, in order absolutely to make the diagnosis you have to look into the bladder with a cystoscope. This disease is predominantly a disease of the female bladder, for in some 104 or 105 cases we have seen only five cases in men. The ulcer is in the dome of the bladder, as a rule, and it requires a long course of treatment to cure it. We feel that about 75 to 80 per cent of them are benefited permanently; some of them are cured. The other 20 or 25 per cent become those persons who come to your office continually and must be treated from time to time. The best treatment is (1) fulguration or desiccation of the ulcer area with the coagulating high frequency electric current, which must be done under anesthesia; (2) while under the anesthesia instill a quantity, perhaps 15 cc. or 20 cc. of a 2 per cent silver nitrate solution, followed in about three minutes by some normal saline in order to precipitate the silver. Sometimes this treatment is supplemented by dilatation of the bladder under pressure, but this latter is not without its dangers, because there have been bladders that have been ruptured in this way, so we are not enthusiastic about using the dilatation for the treatment of Hunner's ulcer, believing that we get better results in other ways.

We have one patient here today. This gentleman is a private patient of Dr. Harry Culver, and the other patients who were clinic patients didn't have time to come over. This man came over to show his gratefulness for the treatment that has been carried out on him.

This gentleman is thirty-five years old and is a chemist. He was first seen by Dr. Culver on May 6, 1943. He began to have pain in his lower back in 1941. About eighteen months later this was coupled with pain in his legs which became to be very severe about the time he went to bed, and he usually suffered all night.

In April, 1943, about a month before he was seen, he began to have an incontinence of urine, an incontinence of overflow, apparently, coupled with the loss of feces, that is, a relaxation of the bowel. At that time his doctors were trying to determine the source of the disorder, whether it was a tabes, whether it was due to a spinal cord tumor, or to what the disease might be due.

His knee reflexes were always normal, so that ruled out tabes and an upper cord tumor. There was only paralysis of his perineum in a definite area of demarcation. He had so much fever that his doctors could do nothing with him until they drained his bladder by cystotomy. When this was done his fever stopped. Then his doctors were able to go ahead and explore the spine to find out where the tumor might be. The tumor was located in the cauda equina down over the sacral region. The tumor which was removed at operation was very much macerated but the pathologist determined that it was probably a dermoid.

He still has some of his untoward symptoms, but he is much better in that his legs are stronger, they no longer give him great pain that he once had. His bowels have recovered. He is still on the cystotomy drain and may have to be on it for a long time yet. He says sometimes when he goes a long time with the tube clamped he overflows his urine down through the urethra.

These cases of paralysis of the bladder come, of course, from tumors, from disease, from trauma. They pose a question in management. There are schools that say never introduce an instrument or catheter into these patients because you will introduce some infection. That has largely become obsolete because of the fact that we have our sulfa drugs which prevent to a large extent infection of the urinary tract. Again, there are other cases which will not develop the so-called automatic bladder which the neurologists all look for, and so something has to be done. One cannot keep a patient on catheter drainage forever, so a cystotomy is done, and the cystotomy in this case was a life saver for it brought the man's temperature to normal, decompressed his urinary tract so that it functioned properly while other measures were being taken. Ultimately this man may recover so that he will get along without a cystotomy tube.

BILHARZ CYSTITIS

There is one other disease which I wish to speak of because here in this audience there are men and women who have sons and daughters, brothers and sisters over in North Africa, in Egypt, and in the Mediterranean area, where bilharzic cystitis is endemic. Two weeks ago I spoke before the Kansas City Southwest Clinical Society and I spoke of bilharzic cystitis, stating that it was now a very important disease. Perhaps some of you have never heard of it. If you haven't, you should go home and read about it because there is a considerable literature about it. It was called to my attention by an Army officer who was in my audience in Kansas City that it is very important. When I had finished speaking he got up and said, "Gentlemen, this disease which Dr. Ockerblad has called to your attention is of extreme importance." He had just come from Egypt. "We had a young man who was ill in the hospital, and he became gravely ill and had a swelling of his parotid glands which were thought to be mumps. However, nothing that was done for the patient seemed to do any good and he finally died. The autopsy showed that every fluid in his body, his spinal fluid, his peritoneal fluid, his blood, and his urine, was filled with bilharzic flukes."

Let me explain to you briefly about this disease. It is endemic in certain Mediterranean countries and in South Africa. It is caused by a blood fluke which was discovered by Theodor Bilharz in 1851. This scientist went down into Egypt and studied the cause of the disease. The reason he found it so readily was because the fluke was in the blood and he examined the blood and urine and found the ova in the urine and the fluke in the blood. The male fluke is a large organism about an eighth of an inch long. The female is much smaller. This, of course, was a long time before Pasteur's time. It was a long time before Lister got interested in infections, and it was a remarkable discovery. Nothing much was done about it for many years. No cure was known for this disease until the Bilharz Commission headed by Lieper went down into Egypt in 1915 to study the disease. They made the important discovery that the fluke had an asexual cycle and a sexual cycle and that the asexual cycle took place in a snail which fortunately does not live anywhere but in that neighborhood. When individuals who have this cystitis pass the ova out through the urinary stream into the ponds or wherever they may happen to be and the snails pick them up the ova pass to the digestive tract of the snails a certain cycle of development goes on, they are extruded in six weeks as cercariae which swim around in the water. When an individual goes wading or gets wet in that part of the country these cercariae stick to the skin and within twenty-eight hours penetrate the skin and get into the venules and by following the venous channels get into the lungs and from thence they go to the portal circulation where they become male and female and their sexual cycle begins. The female then climbs down through the veins to the tiny venules of the bladder and lays her eggs under the mucosa of the bladder.

The importance of the whole thing is this: that our men are over there and that we are going to see, after the war, some of these infections carried over into this country. It is fortunate indeed that the Lieper Commission discovered not only the sexual and asexual cycle but also what to do for it. They discovered that potassium and antimony tartrate given intravenously was a specific, and then of course it remained for others in 1931 to develop a benzene ring compound or a fixed potassium antimony tartrate which could be given more safely than the potassium antimony tartrate of the ordinary type. This was called fuadin, named after King Fuad of Egypt. It is available in ampules. It is a specific for the disease. If copper sulphate could be put where the snails live and fuadin given to all the human victims, this disease could be eliminated from the earth. However, it is to be remembered that in 1934 the special hospitals in Egypt took care of 250,000 of these victims in one year.

There are many other diseases of the bladder which we shall leave for another time.

DIAGNOSTIC MEDICAL CLINIC

The Heart and Hyperthyroidism

By LEWIS M. HURXTHAL, M. D.
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We have three patients to present who will show the effect of the thyroid intoxication on the heart. It is well to remember that the heart in hyperthyroidism is overactivated, so to speak, first because of the increase in metabolism which increases the circulation rate, and secondly, because the thyroid hormone itself is working in the heart increasing the metabolism of the heart muscle, so that in hyperthyroidism the heart is more overactive than one can explain on metabolism alone. For example, if one walks down the street he may increase his metabolism 25 to 50 per cent and yet there is no pounding of the heart, such as one would expect in hyperthyroidism with the same elevation of metabolism. This overactivity of the heart is helpful in diagnosis. The average patient with an irritable heart or neurocirculatory asthenia does not show on clinical examination such an overacting or pounding heart.

There are one or two heart diseases, however, which may mislead one in regard to this simple test of palpating the heart. The first is mitral stenosis, which characteristically is a pounding, heaving impulse. The murmur often is missed in mitral stenosis because the stethoscope is not placed out far enough or because the patient has not been turned on his left side, which often brings out the mitral diastolic murmur, thus differentiating it from hyperthyroidism.

Patients with simple goiter and mitral stenosis are often referred for hyperthyroidism particularly, because in most cases with mitral stenosis fibrillation develops. About 10 per cent of all patients with hyperthyroidism sooner or later develop fibrillation. Half the time it is only transient, but in about 5 per cent it becomes established. Fibrillation is an inefficient mechanism and is one reason why the heart fails from hyperthyroidism. Only a small percentage of patients with hyperthyroidism who develop congestive heart failure have normal rhythm, and when normal rhythm is present it is usually in the patient who has rheumatic heart disease or is past fifty years of age, in whom the myocardium cannot keep up to circulatory demands.

This first patient is a boy with typical exophthalmic goiter who was operated upon in August. He has at the present time a recurrence of hyperthyroidism. His exophthalmos persists. He has a rapid pulse, and on the left side one can feel recurrent thyroid remnants, hyperplastic remnants.

I would judge that his metabolism is not elevated to a great degree at the present time, and yet in spite of this he has a very overactive heart, part of which of course might be due to his being here. This is about the only cardiac finding, it being the simple, overactive heart in hyperthyroidism.

His metabolism on September 9, 1943 was +17 per cent, shortly after operation it was —5, and at the present time it is +25. Twenty-five per cent elevation in the metabolic rate is not excessive and should not cause the pounding heart which he shows readily on palpation, unless it is due to the hyperthyroidism which he has.

The next patient is one step further in hyperthyroidism in its effect on the heart. No one would fail to recognize exophthalmic goiter in this patient. She was operated on in 1940 for exophthalmic goiter, and although better for some time, she recently has lost 30 pounds. At the present time the heart is fibrillating. Auricular fibrillation may occur without great toxicity. As a matter of fact, it is not always a reliable index of toxicity. Some patients develop fibrillation with recurrence without any great elevation of metabolism and without marked clinical symptoms of hyperthyroidism, but the average patient with fibrillation and hyperthyroidism is very toxic.

This patient, too, demonstrates a palpable thyroid remnant on the left side, which is easily larger than many patients with exophthalmic goiter have before the first operation. This patient is waiting for a hospital bed. She has not developed congestive heart failure. From 20 to 50 per cent of patients with fibrillation develop congestive failure.

One of the interesting things about exophthalmic goiter is why it recurs. Some people believe that the recurrence of hyperthyroidism is due to inadequate removal of thyroid tissue. This may be true in some cases, but there are patients (and we have seen plenty of them) who continue to have recurrent hyperthyroidism over a period of fifteen to twenty years, in spite of roentgenologic treatment and operation.

In summary, then, this last patient has the classical symptoms of hyperthyroidism. She feels warm and sweats, has a tremor, palpitation, nervousness, exophthalmos, and a large hyperplastic thyroid remnant, with an overactive heart which is fibrillating.

The next patient, 61 years of age, complains of nervousness and shortness of breath. Her pulse is rapid, without a great deal of overactivity of the heart. As a matter of fact, the pulse is well over 100 at the present moment, without any heart thrusts that can be detected on putting the hand over the heart. The pulse is regular. Nothing can be felt in the thyroid. It may be a little enlarged, but it is not hyperplastic and not adenomatous.

This patient's appetite is variable. She does not complain of heat. She has lost weight during the last four or five years. When she walks or otherwise exerts herself, she has substernal distress which disappears shortly after the exertion ceases.

A record of her metabolism shows extreme variability. This is always against hyperthyroidism. Patients with hyperthyroidism run fairly consistently elevated metabolic rates, not varying, for example, from +40 per cent one day to +10 the next and then back to +35. There are no eye signs or tremor, and the skin is not excessively warm, yet she has tachycardia and substernal distress on exertion, which probably is due at her age to coronary disease.

The question is, does this patient too have hyperthyroidism? Any patient past 50 years of age, with the coronary arteries narrowing, and who can get along in ordinary life without substernal distress, who develops hyperthyroidism or is given thyroid extract, may well develop angina of effort. Patients with coronary disease too are apt to have, when active trouble is going on, tachycardia with gallop rhythm.

I see no record here of an electrocardiographic tracing. That is not important, though. The patient has symptoms which are typical of angina pectoris.

One would be very hesitant to offer this patient surgery for the relief of her symptoms. Iodine could be tried, but probably would not be effective, and I believe the patient's symptoms will have to be controlled by other methods.

It is true that if subtotal thyroidectomy were done, for a period of a month or two there might be marked relief of symptoms due to the enforced rest, the lowering of metabolism from removal of thyroid tissue, but as the thyroid regenerates it will return. Of course, total thyroidectomy might relieve her, but this measure is largely discarded now because of the undesirable effects that occur with the development of myxedema.

I was glad to see this young woman because she happened to be a patient of mine in Boston. She was operated on here, I believe, some ten or fifteen years ago, for typical exophthalmic goiter. She had a recurrence while in Boston, was reoperated and developed postoperative myxedema, for which she is now taking a small amount of thyroid. She is doing well. She had other interesting features in that she was completely hypogonad even before she developed exophthalmic goiter, had never had a menstrual period, and had no pubic hair or breast development. The sella turcica was not enlarged, and her height was normal. At the time of recurrence of hyperthyroidism she showed excessive estrin in the urine, and after operation this continued to be present although in less amount. For six months, no secondary sex changes developed. Then she was given stilbestrol by mouth, following which she is developing pubic hair, enlargement of the breasts, and periodic menstrual bleeding, as is customary on such therapy.

This case brings up an interesting condition, that is, if exophthalmic goiter is the result of stimulation from the pituitary gland, here one part of the pituitary was overactive and the gonadotropic principle was underactive. On the other hand, the presence of estrin in the urine, if one can rely on biologic assays, would suggest that the gonadotropic principle is present, that she also has active ovaries, but the end organs do not react.

We believe it is important to treat exophthalmic goiter and toxic adenomatous goiter early because fibrillation eventually may develop and with that, congestive heart failure. These complications can be controlled, but according to our experience, the mortality rate in patients with fibrillation, particularly those who have had congestive heart failure, is much higher. It may be that with the introduction of Thiouracil, the mortality can be decreased considerably by giving this drug for a month or so before operation in these very serious cases. We have done this in a few elderly patients with hyperthyroidism and cardiac complications.

Dr. Lahey coined the term "thyrocardiac," which has been rather loosely used in describing patients with hyperthyroidism who happened to have some heart disease. As a matter of fact, a patient with hyperthyroidism can have any type of heart disease independent of the thyroid condition. We have limited the term "thyrocardiac" to patients who have hyperthyroidism and who have fibrillation, or to patients with hyperthyroidism who have congestive heart failure, or to both. A thyrocardiac is not a patient with hyperthyroidism and mitral stenosis compensated. A thyrocardiac is not a patient with angina pectoris and hyperthyroidism. In other words, we have limited the term to those patients who develop congestive heart failure precipitated by hyperthyroidism or fibrillation.

There are three principles of treatment in the management of these patients. As you know, for many years digitalis was thought harmful to thyrotoxic patients. There is no doubt that it does good in preparing these patients for surgery. It does seem to be true that it is more effective when fibrillation is present in hyperthyroidism when the patients have coincident heart disease. For instance, if they have rheumatic heart disease, one can slow the pulse much more dramatically than when they have hyperthyroidism and fibrillation without heart disease.

The three principles of treatment are: First, to try to cut down the amount of thyroid toxicity. This in itself may relieve all symptoms of heart failure. Secondly, to try to increase the cardiac function, and this can be done by digitalization if fibrillation is present. Third, to relieve the embarrassing loads on the heart, aside from the hyperthyroidism, such as the accumulation of fluids, which can be done if necessary by diuretics; if not, by tapping.

The reason the heart fails in hyperthyroidism is that it cannot keep up to an abnormal load. If the average patient with heart failure who has no thyroid disease goes into heart failure it is because the organ cannot keep up to the normal load or the normal metabolism. But in a patient with hyperthyroidism and congestive heart failure, the heart fails because it cannot keep up to an abnormal load. Ordinarily in a patient with congestive heart failure without hyperthyroidism, the circulation rate is very slow as done by the various tests, but the patient with hyperthyroidism and congestive heart failure may be found to have normal circulation rates. If they did not have heart failure they should have an abnormally rapid circulation rate, so that congestive heart failure can be present in a patient with hyperthyroidism with an essentially normal circulation rate but not normal for the load it has to carry, therefore the heart fails.

Apparently hyperthyroidism does not cause cardiac enlargement. The heart is dilated as it is in all patients who have congestive heart failure. These roentgenograms (slide) demonstrate the shape and size of the heart in a patient with exophthalmic goiter and fibrillation, showing the prominence in the region of the pulmonary artery and also the left auricle, and the general dilatation. This patient had congestive heart failure, as we recognize it clinically, with pitting edema, enlargement of the liver, and engorgement of the neck veins. In the next slide, one sees the reduction in the size of the heart after hyperthyroidism has been relieved.

(Slide) Here is an example of a patient with severe exophthalmic goiter of seventeen years' duration, who was so ill with congestive heart failure that he was cyanotic and pulseless. From various hospital records for a period of seventeen years, we know that he had exophthalmic goiter. He had a large hyperplastic gland. This picture was taken almost eighteen years ago, and the roentgenologist at that time said this was pericardial effusion. (Slide) After the first operation and when he had been rid of congestive heart failure, you can see the heart is without any doubt of normal size.

(Slide) Here is another example of the change in size of the heart without a change in rhythm. Often when the fibrillation is relieved after operation, there is a greater reduction in size than when fibrillation persists. In the first instance, congestive heart failure was present with fibrillation; in the second instance, fibrillation persisted without heart failure.

(Slide) These are tracings of superimposed plates showing what happens during congestive heart failure. Congestive heart failure in hyperthyroidism from the physical point of view is no different from any other congestive failure, and it shows that the diaphragm is high when the patient has congestive heart failure due to the turgidity of the lungs and enlargement of the liver which pushes up and also displaces the heart a little to the side; dilatation of the auricles, and the subsequent lowering of the diaphragm at various stages after relief of heart failure before operation; and after a second operation three months later, gradual decrease in the size of the heart which is, as I said, due probably to dilatation.

(Slide) It was interesting in going over a group of roentgenograms on patients with hyperthyroidism who had had the disease for over four years and then going over a group on patients who had had the disease for less than six months, matching age for age and sex for sex in a group of about 50, to find that there was no difference in the size of the heart between those who had had hyperthyroidism for a long time and those who had had it for a short time.

In regard to the principles of treatment, as I have previously stated, two things are to be accomplished: One is to reduce the degree of hyperthyroidism, and the other is to increase circulatory efficiency. Here is a chart of a man who had exophthalmic goiter with congestive failure and normal rhythm, who was given only iodine but no complete bed rest and was allowed to be up and around; he had a diuresis on the administration of iodine which by lowering the hyperthyroidism allowed his heart to compensate.

(Slide) This is a chart of the man who I said was pulseless at the time of admission with exophthalmic goiter, who was given intravenous digitalis to start with. His pulse became perceptible the next day and his blood pressure was also obtained the next day. When he was given digitalis and bed rest, his weight decreased about 5 pounds and then remained stationary on digitalis until Lugol's solution was given. This demonstrates, I believe, although I cannot prove it, that digitalis had reached its maximum effect when his weight remained stationary and then when Lugol's solution was given the hyperthyroidism decreased, allowing the heart to work with less of a burden, following which he had a further diuresis on iodine alone.

(Slide) Another example of the effectiveness of digitalis in a patient with hyperthyroidism who was allowed to remain in bed without treatment, gaining 8 pounds from increasing edema during the week in the hospital; then he was digitalized within a period of a few days, producing a drop in weight with diuresis and slowing of the apex and apical rate; then his condition remained more or less stationary until diuretics were necessary to produce further diuresis. This chart was taken some years ago when we were using theocin with good results.

Here is another example of the effectiveness of digitalis without the administration of Lugol's. This hospital chart shows a probable error in taking the pulse, but at any rate the weights are the important thing. She remained in bed four days and then was given a large dose of digitalis with a rapid diuresis, which, of course, could have come spontaneously on four days of rest, and then the administration of Lugol's solution after a few weeks with a gain of apparently real weight.

This next chart shows the increase in weight without treatment for four days and then the administration of digitalis, showing the effect on the pulse rate and a prompt diuresis, followed by further diuresis with a diuretic some days later.

We have felt that it was desirable to try to stop fibrillation in patients after all surgical procedures were carried out, and for this reason we have given quinidine more or less routinely unless there were contraindications. However, from the follow-up of our patients, it is doubtful whether quinidine really has accomplished anything worth while. It is true that it does stop fibrillation, but in all probability a good many of these patients would have stopped fibrillating some months after operation, without quinidine. We know that before quinidine was given, figures reported in the literature indicated that about 50 per cent of these patients stopped fibrillating spontaneously. Following all our cases from 1922 to 1943, over 600 in number, we find at the present time that of those patients living, 28 per cent have fibrillation.

(Slide) If we felt that the persistence of fibrillation was a dangerous thing, we would expect to find many more deaths in those who had had fibrillation for a long time, but in those operated in the years 1922 to 1931, 27 per cent are fibrillating; in the group operated between 1931 to 1936, 25 per cent; 1937 to 1941, 28 per cent. This shows there were no more deaths among the patients who continued to have persistent fibrillation a long time than those who had it a short time. In other words, it does not appear that the persistence of auricular fibrillation is dangerous. It is true a good many of these patients have taken digitalis regularly after operation, but this fact should not diminish the possibility of embolism although it would reduce the chance of congestive heart failure.

THE SPEAKER: At this time I have a little business to take up with you and report the nominations for officers.

For President:
  Brigadier General Fred W. Rankin,
  Washington, D. C.
President-Elect:
  Donald Balfour, Rochester, Minnesota.
Presidents of Clinics:
  Edward W. Archibald, Montreal, Quebec, Canada.
  John F. Erdmann, New York, New York.
Managing Director:
  Arthur G. Sullivan, Madison, Wisconsin.
Secretary:
  Tom B. Throckmorton, Des Moines, Iowa.
Treasurer and Director of the Foundation Fund:
  Henry G. Langworthy, Dubuque, Iowa.
Speaker of the Assembly:
  Mather Pfeiffenberger, Alton, Illinois.

What is your pleasure? Will these be your officers next year? All in favor say "aye"; opposed "no." The "ayes" have it.

For Board of Trustees:

Henry G. Langworthy, Dubuque, Iowa.
John E. O'Keefe, Waterloo, Iowa.
Tom B. Throckmorton, Des Moines, Iowa.
Mather Pfeiffenberger, Alton, Illinois.
George V. I. Brown, Milwaukee, Wisconsin.
John M. Dodd, Ashland, Wisconsin.
Eugene S. Sullivan, Madison, Wisconsin.

What will you do with those, elect them? All in favor say "aye"; opposed "no." The "ayes" have it and they are elected.

* * *

At this time I do want to take the opportunity again to thank the Medical profession of Chicago and the Palmer House management for making this meeting such a success. I feel, as most of you have expressed to me, that this has been a very, very successful meeting. It has been so successful and we have liked our hosts so much that since they have invited us to come back next year we have accepted their invitation for the third week in October of next year.

The Board of Directors want to thank you also, and we welcome your constructive criticism.

* * *

Dr. Gradle, our next essayist, was called unexpectedly to Washington. As you know, he is one of those busy men who is giving of his time to our national cause.

Treatment of Eye Injuries by the Man in General Practice, the next address will be presented by Dr. Samuel J. Meyer, Attending Ophthalmologist, Michael Reese Hospital and Illinois Eye and Ear Infirmary.

DIAGNOSTIC OPHTHALMOLOGICAL CLINIC

Treatment of Eye Injuries by the Man in General Practice

By SAMUEL J. MEYER, M. D.
Attending Ophthalmologist, Michael Reese Hospital and Illinois Eye and Ear Infirmary, Chicago, Illinois

That the war has resulted in the realization of the need of greater knowledge on the proper care of the wounded is foremost in the minds of all medical men. Our interest in traumatic surgery has been greatly awakened. Judging from the experience of our British colleagues, ocular injuries occur with surprising frequency as the result of air raids. For example, during the fall of 1940, one London ophthalmic hospital treated 250 cases of ocular injuries (including foreign bodies) in a single night. Our increased rate of production also will add its share to the estimated 315,000 eye injuries occurring annually in this country. The National Health Survey indicated that there are 425,000 persons in the United States blind in one eye. Of the cases studied, 51.9 per cent were caused by accidents, 19.1 per cent by occupational accidents. According to research, the total compensation cost to industry itself of these preventable injuries is believed to be more than $100,000,000, but only 20 per cent or 60,000 of these eye injuries are compensable, and it is estimated that the cost of the 240,000 so-called trivial eye injuries is more than $10,000,000 a year. Furthermore, it is said that accidents account for one-fifth of the entire amount of blindness. With these thoughts in mind, I feel the treatment of ocular injuries would be a suitable topic for discussion.

INJURIES OF THE EYELIDS

Such injuries may take the form of contusions, tears and burns. Contusions are best treated by rest and cold compresses of boric acid or of lead and opium lotion. Incised and lacerated wounds of the lids are fairly common and are frequently extensive. In their repair, one must be guided by the principles of fresh wound healing, which will be briefly reviewed.

1. *Haemostasis.* The ideal control of hemorrhage neither permits the development of hematoma nor justifies the use of necessary or mass ligatures, nor dressing tight enough to starve injured tissues.

When there is hemorrhage in a wound, the blood acts as a foreign body and should be removed because it distends living tissue, hinders the normal blood supply of cells and encourages the development of bacteria.

2. *Infection.* Carefully cleanse and sterilize skin surfaces. Use strict aseptic technique in repair. Fresh wounds prior to the lapse of 6 or 8 hours, may contain bacteria as a surface contamination, but these have not yet invaded living tissues. Efforts to kill bacteria in a fresh wound may not only be unsuccessful but may do unwarranted harm to living cells. Bacteria are thus best removed by thorough irrigation with normal salt solution. Bacteria feed upon dead or devitalized cells, hence damaged tissue should be cut away to prevent necrosis. Normal tissue, if damaged by rough handling, improper ligatures and sutures, or strong antiseptics will become dead tissue.

3. Severed tissues should be replaced and maintained in a position to stimulate normal anatomic relations as far as possible.

4. Nutrition of the wounded tissues should be restored and conserved. Preserve the blood supply, avoid mass ligatures, use fine hemostats, needles and ligatures, tie sutures loosely to allow for swelling, and avoid tension in sutures.

5. The patient and the repaired wound should be placed at rest, with due attention to the general comfort of the patient and to the support or immobilization of the wounded part.

To be more specific, the following technique has been found quite satisfactory for eyes:

Cleanse skin thoroughly with soap and sterile water, clip lashes and shave eyebrow if involved. Follow this by painting the surgical field with tincture of merthiolate. Irrigate the wound with warm sterile saline solution, or, if a dirty wound, equal parts of saline and peroxide of hydrogen, used in an ear syringe, is preferable. This not only removes foreign matter and blood clots, but acts as a hemostatic as well. Use chromic catgut, triple-0 for buried sutures. Use fine silk, four-0 or five-0, in the conjunctiva and skin, taking care not to make the sutures too tight, nor the flaps under too much tension.

Lacerations may be broadly classified into horizontal and vertical. The horizontal one takes the general direction of the fibers of the orbicularis palpebrarum muscle and does not tend to gape. It usually heals without appreciable scar formation and requires few if any sutures. Vertical wounds tend to gape widely from the traction of the orbicularis muscle and, if serious deformity of the lid is to be avoided, careful attention must be given. Primary suture is the best treatment and accurate apposition of the lid margins must be secured. Otherwise, an artificial coloboma or notch will result. The technique used to repair this type of wound is a surgical problem worthy of detailed description.

The halving procedure as described by Wheeler is the operation of choice. The important point in this operation is that the tarsal incisions and skin incisions should never be in the same position, but should be made in such a way that there is overlapping. The amount of tarsus excised should be as great at the upper end of the incision as at the lower. This tends to produce a temporary over-correction of the defect which guards against a recurrence of the notch.

Another frequent type of injury is a laceration or tear of the lower lid near the inner canthus. These wounds usually result in a bad deformity with a destruction of the lower canaliculus. The inner end of the lower lid is carried down and away from the globe, leaving a small space in which the tears accumulate. Repair of this type of injury calls for proper suturing of the lower lid to maintain its contact with the globe. This can only be secured by carrying the lid flap up and well back. The tip must be relieved of all tension even if a canthatomy has to be performed. In the late repair of this type of deformity, all scar tissue must be excised and a sliding flap extending well beyond the outer canthus used to insure ample release of tension on the tip. Preservation of function of the lower canaliculus should be attempted by uniting its cut end to the lacrimal sac. Patency of this canal can sometimes be established by the use of a thread passed through the canaliculus into the lacrimal duct and out through the nose. This suture is left in place for two to three weeks and its position is shifted daily. Perforating wounds of the lids are not in themselves serious unless some of the deeper structures are injured, since these wounds usually heal without becoming infected.

In all cases of injury to the lids, the possibility of underlying fracture should be considered and excluded by careful palpation and X-rays. Emphysema of the lid indicates rupture of the nasal air cells and should be treated as a contusion with special avoidance of blowing the nose. A distressing sequela of fractures of the floor of the orbit is an enophthalmos which often does not make its appearance until several weeks after the accident. In a series of such cases, X-ray examination has shown a fracture of the floor of the orbit with herniation of the orbital fat in the antrum.

Burns of the eyelids constitute one of our most serious problems. First degree burns heal without difficulty, but second and third degree burns produce severe cicatrization with all its attendant problems. The immediate care of severe burns is that of protection of the globe. Non-irritating oils and ointments are indicated. The final care will depend upon the sequelae. If the ectropion is not too extensive, it can be corrected by a free graft from the upper lid of the contralateral side. Often the severity of the accompanying facial burns precludes the use of any but epidermal grafts.

INJURIES OF THE GLOBE

Contusions of the globe are often followed by intraocular hemorrhage and should be treated with complete rest, cold compresses and frequent instillations of atropine sulphate solution 1 per cent to guard against the development of a traumatic iritis. The treatment of the more serious complications of dislocation of the lens, commotio retinal and detachment of the retina, etc., are not within the realm of this discussion. Conjunctival lacerations often heal spontaneously and require suturing only if there is a wide separation of the edges of the wound. Subconjunctival hemorrhage should be treated with cold compresses for the first 48 hours, after which time absorption of the blood may be hastened by the use of heat. Conjunctival foreign bodies usually lodge under the upper lid and their removal is easily affected after eversion of the upper lid. In the search for a foreign body, it is well to remember that a cilium may become lodged into the upper or lower punctum and be readily overlooked.

The most common type of eye injury is a foreign body embedded in the cornea. It is an experience we have all had and one which every physician is forced to treat at some time. The two essentials in the management of such a case are: first, good illumination, and, second, local anesthesia. Concentrated light which can be shifted across the cornea is essential for the detection of small particles, as is sufficient magnification to assist those physicians of presbyopic age. A bright hand flash light and a binocular Berger loupe can serve well. A local anesthetic to be recommended in place of cocaine is pontocaine hydrochloride one-half per cent, a drop or two of which produces a rapid and satisfactory anesthesia. Another valuable drug is fluorescein. A drop of a one per cent aqueous solution of fluorescein stains any break in the conjunctival or corneal epithelium a brilliant green. Its use should be routine after the removal of a corneal foreign body, because any break in the corneal epithelium may, if neglected, lead to serious complications. It is my practice to protect all eyes with corneal abrasions until no staining exists. Such a case receives a liberal amount of 5 per cent sulfathiazole ointment into the lower cul de sac and a firm protective dressing. In my opinion, the continued use of a local anesthetic without a dressing predisposes the eye to infection, the one complication we all fear in a corneal foreign body. The treatment of corneal ulcers is too technical to permit of discussion here, but those of us who encounter these cases realize the importance of prophylaxis.

The immediate removal of multiple foreign bodies in the cornea is sometimes unwise, for the manipulation necessary for their removal may do more harm than good. A protective dressing and removal of the more prominent ones is often sufficient.

Many foreign bodies strike the eye with sufficient force to penetrate the globe. Never forget that a small corneal laceration may have been the site of entrance of a foreign body. An absent or shallow anterior chamber with a reduction in the intraocular pressure are definite signs of perforation. The history is often misleading, for one must bear in mind that these accidents occur so quickly that the patient does not realize what struck him. In all such suspicious cases, a protective dressing and prompt X-ray examination are indicated. Recent refinements in X-ray technique often enable us to disclose even fragments of glass within the globe. All cases of intraocular foreign bodies require the care of a competent roentgenologist and ophthalmologist. Penetrating wounds of the globe of any kind should receive similar treatment. According to Joy's studies, sympathetic ophthalmia occurs in 5 per cent of all cases of penetrating wounds of the eyeball. The treatment of this disease, once it has arisen, is most discouraging, so every physician should be aware of its possibility and cooperate with the attending ophthalmologist in its prevention. This usually means the prompt removal of hopelessly injured eyes, as well as those with extensive lacerations in the region of the ciliary body.

The treatment of burns of the conjunctiva and cornea is extremely unsatisfactory. The immediate care of chemical burns should consist of copious irrigation with any clean non-irritating solution, care being taken that the folds are well everted so that the whole surface of the conjunctiva is thoroughly cleansed, and no particles or other chemicals remain adherent to it. The dispatch with which such irrigation is used is more important than the kind of solution employed. Attempts at neutralization of the chemicals are usually unsuccessful. Burns with sulphur dioxide and other refrigerants are best treated with repeated instillations of olive oil as these chemicals are usually insoluble in water. Mustard gas is active only in the presence of water, so should be treated with a bland oil. Thies recommends for severe burns of the conjunctiva, immediate grafts of mucous membrane after thorough excision of the necrotic conjunctiva. It is a suggestion worthy of serious consideration. Among the sequelae of severe burns are adhesions between the lids and globe (symblepharon), extensive corneal opacification with vascularization and secondary glaucoma. The management of all these conditions is almost hopeless. Corneal transplantation is usually not satisfactory in the presence of highly vascularized cicatricial tissue such as is seen in old chemical burns.

REMOTE TRAUMA

Remote trauma to the skull may easily produce ocular changes. In skull fracture accompanied by hemorrhage, the increased intracranial pressure not infrequently is manifest in the optic nerve head, as well as by ocular palsies. If the fracture involves the optic foramen or the orbit itself, hemorrhage into the orbit, or edema of the orbital contents, may produce an exophthalmos, an ophthalmoplegia, either internal or external or both, a rigid pupil, and loss of sight. The recovery from these consequences depends upon the rapidity of absorption of the pressure producing fluid, and whether or not such fluid lies between the meningeal sheaths of the optic nerve. Frequently, an old basal fracture that involved the optic foramen and that occurred months to years previously, can be diagnosed by the condition of the optic nerve head. There is no local treatment for such ocular complications of skull fractures.

The New Therapy of Common Skin Diseases

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An elementary classification of diseases of the skin might divide them into those that are curable and those that are incurable. Unfortunately, the belief has become widespread among many of the laity and even among some members of the medical profession that the majority of skin diseases are incurable, an idea that has led to indifference when it comes to their treatment. An analysis of skin cases seen in the office of a dermatologist indicates that from 60 to 75 per cent (and perhaps even a greater number) come under one of the following ten groups: (1) eczema (including dermatitis venenata), (2) acne (vulgaris and rosacea), (3) scabies, (4) ringworm, (5) seborrhea, (6) impetigo, (7) psoriasis, (8) urticaria, (9) warts, and (10) skin tumors (nevi and malignancies). Since the turn of the century remarkable progress has been made in the treatment of diseases of the skin and numerous of the above named conditions which were regarded as incurable or as responding poorly to treatment are now either curable or controllable to such an extent that the affected individual need not suffer. The following review covers the most recent ideas concerning the therapy of these conditions as well as others commonly seen by the general practitioner.

ECZEMA AND DERMATITIS VENENATA

Most difficult of the ten groups of skin diseases listed above to discuss is eczema, which is a term still applied to certain characteristic manifestations in the skin which may be of exogenous or endogenous origin, and localized or generalized. In fact the subject is entirely too broad to possibly consider in the alloted time the various therapeutic measures applicable to the many types of eczema or dermatitis. We know, however, that except for the atopic type which is associated with an allergic family history, virtually all types are responsive to treatment if the cause is removed, and sufficient ingenuity is employed in preventive measures and therapy. The housewives who react to soap (and the number is on the increase with a deterioration in the quality of soaps and the lack of domestic help due to the war) cannot be given new and soap resistant skins but their eczema is certainly curable if they can be taught methods of avoiding irritating contacts. The recognition of cosmetics, particularly nail polish and hair shellac as common irritants during the past few years has solved many cases diagnosed as eczema. After removal of the cause the mildest skin remedy suffices to cure. Improvement in the means of detecting etiological factors by means of percutaneous, intracutaneous and patch tests, often leads to the elimination of the cause and decreases the likelihood of further attacks. In the atopic types of eczema with a family history of other allergic diseases such as asthma or hayfever, cure can never be promised and measures must be directed toward keeping the skin in the best possible condition. Proper vitamin therapy is one of the newer ideas concerning the therapy of eczema and I point to the studies of Paul Gross who found 18 of 24 patients with nummular eczema responded well to vitamin A in doses of from 30,000 to 75,000 U.S.P. units daily for therapeutic purposes and followed by maintenance doses of 25,000 U.S.P. units daily. In infantile eczema the development of soybean milk as a substitute for cow's milk has contributed much to the successful treatment of many otherwise resistant cases.

ACNE (VULGARIS AND ROSACEA)

Acne vulgaris is perhaps the most neglected of all skin diseases and many times because of an indifferent attitude on the part of the family physician. In adolescents one cannot speak of cure as the disease is a developmental process and the underlying cause cannot be stopped but I believe that all cases properly handled may be controlled and the child reach maturity with little or no scarring and pitting. Of greater importance than the lifelong cosmetic disfigurement caused by a severe acne is the mental anguish and often persistent inferiority complex caused by the condition which the affected individual knows is readily apparent to everyone. The modern day treatment of acne vulgaris requires:

1. Consideration of development changes and endocrine factors. Measures must be taken to see that menstrual function is regular.

2. A dietary regime with the elimination of excess fats and sweets.

3. Attention to the proper functioning of the gastrointestinal tract.

4. Attention to the general health (exercise, tonics, Vitamin D).

5. Proper hygiene of the skin. (In the majority of cases this means the frequent and thorough use of soap and water and removal of comedones, preferably by the physician.)

6. Control of local infection (sulphur or mercurial lotions or pastes).

7. Control of overactive sebaceous glands (X-ray therapy properly administered is perfectly safe and in resistant cases the only method of controlling the disease).

8. Attention to the scalp (an oily or scaly scalp is a source of aggravation in acne).

Vaccine therapy, so widely used, is perhaps the least efficacious therapeutic procedure that can be used, as the most it can possibly do is to lessen the tendency to secondary infection.

Unsightly scars resulting from a severe and often neglected acne can now be lessened by the repeated application of a mixture of carbon dioxide snow, precipitated sulphur and acetone applied at intervals of one to two weeks, but are never entirely removed.

Acne rosacea, which may be due to a variety of causes, particularly digestive disturbances, improper diet, nervous excitation and menopausal disturbances, has been found to often respond to riboflavin in doses of 3 to 5 mg. daily. Rosacea keratitis in particular improves strikingly with riboflavin administration.

SCABIES

Scabies, long cured chiefly with sulphur, has recently been treated with newer and more elegant methods including (1) sodium thiosulphate, plus dilute hydrochloric acid; (2) benzyl benzoate; and (3) rotenone. According to recent studies by Ingels, the safety margin of the benzyl-benzoate cure has a great advantage over the sodium thiosulphate cure and consists of rubbing into the skin with a soft brush a solution of equal parts of benzyl-benzoate, sapomollis and iso-propyl alcohol.

RINGWORM

The type of ringworm infection seen commonly today is that involving the hands and feet. Early recognition and proper treatment of this infection may prevent the development of one or several complications which include: (1) the development of a generalized rash or "dermatophytid," (2) lymphadenitis, (3) cellulitis, (4) phlebitis, and (5) erysipelas. While all types, except that caused by the Trichophyton purpureum may be regarded as curable, no single cure or cure-all has ever been found, therapy varying according to the type and stage of the infection.

A. *Vesiculo-bullous* (acute type).

1. Incision of all lesions to promote outward drainage.

2. Foot baths or continuous wet dressings of potassium permanganate (1-4000 solution), saturated boric acid solution or liquor aluminum acetate (Burow's Solution).

3. Avoidance of all strong applications such as Whitfield's Ointment.

B. *Dry squamous and intertriginous type* (usually secondary to vesiculo-bullous).

1. X-ray therapy: one or several fractional doses.

2. Castellani's paint.

3. Desquamating and fungicidal ointments (one-half to full strength Whitfield's Ointment).

C. *Chronic hyperkeratotic type.*

1. Strong desquamating ointments. An ointment composed of equal parts of salicylic acid and lanolin applied on gauze and bandaged in place for three-day periods often acts miraculously.

Refined proprietary modifications of such simple formulas as Whitfield's Ointment may be employed successfully at times, but one should always know the ingredients and their relative strengths in such preparations and employ them only at the proper time.

Quick and satisfactory cures for ringworm of the scalp or nails have never been found, although both types may be cured over varying lengths of time.

SEBORRHEA

The term seborrhea is used to cover everything from a simple scale or dandruff of the scalp to a severe eczematization involving the scalp, face, ears and even the trunk. It is my personal opinion that seborrhea in its mildest form represents merely the normal desquamation of dead cells from the epidermis, but which under certain conditions may become accelerated with a piling up of the cells and providing a perfect media for growth of certain types of bacteria which are almost universally recovered in the more severe types. Mild types are controlled by good hygiene, but in the more pronounced cases a bactericidal agent is required, sulphur being the old standby. A usually effective application is sulphur and salicylic acid incorporated in one of the newer ointment bases such as aquaphor or qualatum:

℞	Sulphur ppt.	ʒ 1
	Acidi salicylici	ʒ ss
	Aquaphor	℥ ii

Applied to the scalp nightly or less often and followed in the morning by a shampoo, seborrhea soon disappears and may be kept under strict control by an occasional application. Recent studies indicate that seborrhea is favored by a deficiency of vitamin B and possibly narrows down to a deficiency of pyridoxine (vitamin B_6) although this is by no means proven.

IMPETIGO

Recent studies indicate that impetigo is perhaps always caused by staphylococci, rarely if ever by streptococci and responds to the local use of sulfa drugs, particularly sulfathiazole. Sulfathiazole incorporated in 5 to 20 per cent strength in a jelly or vanishing cream base will cure impetigo in from 3 to 9 days and the new microcrystal sulfathiazole is said to occasionally cure in 24 hours. Unfortunately, certain patients are intolerant to sulfa drugs applied locally, while others are apparently sensitized so that they are unable to take sulfa drugs by mouth at a later date. At present enjoying a great wave of popularity, only time will tell whether sulfa drugs will replace older remedies such as ammoniated mercury and gentian violet in the therapy of impetigo.

PSORIASIS

Representing about 6 per cent of all skin conditions, psoriasis is still a disease of unknown etiology and incurable. However, satisfactory therapeutic results are possible in the majority of cases with patient and persistent treatment. To treat psoriasis ideally, hospitalization is necessary where either (1) the Mayo Clinic method of applying crude coal tar salve at night, removing it in the morning and exposing the patient to ultraviolet rays, or (2) the Schamberg method of placing the patient on a low nitrogen diet and applying gradually increasing strengths of salicylic acid, tar and chrysarobin ointments may be resorted to. Following the Schamberg method I have known some patients to remain virtually free of patches for periods as long as ten to twenty years, although earlier relapse is the rule. There are no new methods of treating ambulatory cases that may be considered revolutionary. About 1 in 10 patients will respond to massive doses of vitamin D (100,000 to 300,000 units daily), but the method cannot be considered entirely safe due to the marked rise in blood calcium that follows. Soybean lecithin given in large doses in cookies or wafers has been suggested, but has been disappointing in a number of my patients.

URTICARIA

Only chronic urticaria and angio-neurotic edema may be considered as difficult to cure. Of 35 cases treated by histaminase by Layman at the University of Minnesota, 21 were cured and 14 were unimproved. Histaminase by mouth has not proved of demonstrable value except in those physical allergies where lesions appear as a result of cold, heat, light, etc. The injection of estrogens has proven curative in some cases in women. Many cases of chronic urticaria prove rebellious to the most expert treatment but here not only the dermatologist, but medical science in general fails.

VERRUCAE

The past quarter century has seen the following therapeutic procedures suggested for verrucae: (1) neoarsphenamine intravenously, (2) bismuth intramuscularly and injected directly into the wart, (3) sulfarsphenamine intramuscularly, (4) injection of distilled water, (5) intracutaneous injections of cow's milk; (6) autohemotherapy, (7) vaccines made of ground up warts, and (8) autosuggestion. A simple infection caused by a filterable virus, multiple warts often prove most refractory to treatment, yet like many other infections may at times disappear spontaneously. The best treatment for warts except on the plantar surface is immediate removal of the initial growth before autoinnoculation and multiplication takes place. The best treatment I have found for plantar warts is to remove the excess and covering callus tissue by paring or several days application of a 40 per cent salicylic acid plaster, followed by X-ray or radium therapy in proper dosage. Surgery is the court of last resort, but is at times required for extensive lesions or lesions that have been overtreated by X-ray or radium. Grafting is usually required following removal of growths surgically.

SKIN TUMORS

(Nevi, Keratoses and Skin Carcinoma)

Under this heading could be included a large number of conditions of the skin but discussion here will be limited to nevi and malignancies. Of the nevi often designated as "birth marks" and appearing in the infant, interest centers chiefly around vascular lesions. Port-wine marks remain an unsolved problem but elevated vascular nevi may now be treated by (1) radium, (2) carbon dioxide snow, (3) sclerosing injections, and (4) surgery. Small lesions such as the so-called strawberry marks are best treated by carbon dioxide snow, but larger lesions such as the cavernous angioma respond better to radium or sclerosing injections or a combination of these two therapeutic procedures. Treatment should be instituted as soon as the lesions are recognized as they tend to enlarge as the child grows.

The dermatologist does not recommend that other types of nevi, such as nevus pigmentosus or nevus pilosus necessarily be removed, but all nevi subject to chronic irritation are best eradicated and those of blue-black color should be removed by wide excision because of the danger of melanoma.

There is no excuse today for the development of incurable skin cancer. Any growing persistent lesion of the skin in an elderly patient should be regarded with suspicion and preferably removed and examined microscopically. The development of the method of destroying skin growths by electro-fulguration and their removal with electric cutting currents have advanced to such a degree in the past twenty years that removal of skin growths is today a relatively simple procedure. Also the understanding of the method of application of radium and roentgen rays is greatly advanced and both are at times of great value in the treatment of premalignant and malignant conditions of the skin in expert hands.

OTHER DERMATOSES

Until 1925 lupus erythematosus was considered entirely incurable. Today with bismuth or gold and sodium thiosulphate the majority of cases of the discoid type are curable. Disseminated lupus erythematosus remains incurable and almost invariably fatal. Keratosis follicularis (Dariers disease), a rare cutaneous order, responds at least in part to massive doses of vitamin A. Fissuring at the angles of the mouth once invariably regarded as an infection, responds promptly to riboflavin therapy. The cause and cure of premature idiopathic alopecia remains a mystery, but there has been some encouragement or slight benefit from the administration of one of the fractions of vitamin B. Slowly but surely the list of curable skin diseases is being increased. The day has passed when dermatology should be regarded as a mystic science and the victims of skin diseases regarded as unworthy of the careful attention of their physician. The patient cured of an unsightly skin disease is ever grateful.

. . . Dr. G. V. I. Brown, Milwaukee, took the Chair. . . .

THE CHAIRMAN: *It is my pleasure to introduce Dr. Edward N. Cook, Lt., Medical Corps, Volunteer Specialist, U.S.N.R., Mayo Clinic, Rochester, Minnesota, who will speak on Causes and Treatment of Symptoms Recurring after Transurethral Prostatectomy.*

Causes and Treatment of Symptoms Recurring after Transurethral Prostatectomy

By EDWARD N. COOK, M. D.
Section on Urology, Mayo Clinic, Rochester, Minnesota

As with any operative procedure in the field of medicine, certain symptoms and complaints may occur after transurethral prostatic resection. It is true that some of these postoperative difficulties may be inevitable but many of them are related to technical errors or inadequacy on the part of the surgeon, while others are due to improper postoperative care. A most complete knowledge of the possible causes of these complaints is necessary if the physician or surgeon is to be able to care properly for these problems.

Because transurethral prostatic resection has supplanted other forms of prostatectomy almost completely at the Mayo Clinic, I shall confine my remarks primarily to the group of patients whose prostatism is relieved by transurethral means. However, this does not mean that dysfunction does not occur after prostatectomy performed by either the perineal or the suprapubic method of approach. Thompson and Cabot as well as others called our attention to this fact many years ago, and of great interest to me is the appreciation that while certain postoperative complications may be related particularly to one or the other route of approach to the prostate, nevertheless all of them are related intimately to the same possible causes of trouble. It is these causes of trouble, methods of avoiding them, and methods of treating them if they do occur, which I wish to discuss in detail.

Only too often is the medical man prone to discuss only his good results. The discussion in this paper is not presented because of the frequency of these complaints. As a matter of fact, only a relatively small percentage of patients experience any appreciable distress after operation. In a previous discussion of this subject before our own staff, I called attention to the considerable apprehension that many patients have postoperatively. They have had their difficulty for so long and in many instances expect too much. Frequently their symptoms and complaints are minimal, are present without demonstrable findings and can be relieved by a few words of reassurance.

We are all more or less familiar with the complications which may follow prostatic operations. In the early days of transurethral resection, bleeding and infection headed the list just as they plagued the perineal or suprapubic prostatectomist in the early days of these latter procedures. With increased technical skill on the part of the surgeon these and other early postoperative complications are being kept at a minimum and are observed by no means as frequently after transurethral prostatic resection as after either perineal or suprapubic enucleation of the adenomatous prostate.

Let us discuss the difficulties which occur after transurethral prostatic resection, from the angle of the patient's complaint, as that is the prime motive of this paper. Appreciating the symptoms, we then can discuss the findings and make a few suggestions as to the cause and treatment of each.

NARROWED URINARY STREAM

There are two primary reasons for the complaint of narrowing of the urinary stream. The first of these is entirely technical and rests with the surgeon. Inadequate removal of tissue, which figures so prominently as a cause of distress after operation on the prostate, is all too often responsible for trouble. The inadequacy of the surgeon is usually the answer and incomplete removal of the adenomatous tissue may leave a distressing chain of symptoms. The technic of transurethral prostatic resection must be mastered thoroughly and if the procedure is performed satisfactorily and completely an adequate urinary stream will result. Occasionally, owing to the general condition of the patient or the extreme size of the prostate, the operation may have to be terminated before it is finished. Such being the case, it is important that the patient not be dismissed from the surgeon's care if there is any trouble with the urinary stream.

A second cause of a narrowed stream is postoperative stricture. This infrequently is situated in the posterior urethra, as evidenced by scarring of the vesical lip. This complication occurs more frequently after resections performed with the high frequency loop than with the cold punch resectoscope, and can usually be relieved by dilatation with the Kollmann dilator, and in rare instances by incision with the resectoscope. Anterior urethral strictures are a most distressing complication. If a stricture is present preoperatively, gentle and satisfactory dilatation should be carried out before the resectoscope is passed. Unless the urethra is of a size to take the resectoscope easily regardless of size, perineal urethrotomy should be performed and the instrument passed through this opening. Another point to mention is the care in handling the penis with the resectoscope in place. The surgeon's hand should not grip the instrument around the penis. By observance of this precaution, trauma may be avoided. Narrowing in the region of the fossa navicularis or at the external meatus is a frequent finding. It is my feeling that meatotomy in this last group is of more value than dilatation because there is less scarring in the postoperative healing process. If the tissues have been torn during dilatation, the resulting scar and contraction may present a real problem postoperatively.

If a stricture and periurethritis do develop, the use of hot applications, a small indwelling catheter and, later on, the passage of sounds will relieve the patient's distress. Unfortunately, occasional passage of a sound may be necessary for a period of months.

BLEEDING

Bleeding may be divided into that occurring at the time of operation or immediately after and that occurring late. Excessive loss of blood at the time of operation is a frequent cause of such complications as oliguria, renal insufficiency, phlebitis, pneumonia or secondary anemia. If the patient has any cardiac imbalance, excessive hemorrhage, with the resultant fluctuations of blood pressure, may precipitate a sudden cardiac embarrassment of greater or less degree. Hemostasis at the time of operation must be carried out with extreme care. If done as one proceeds with the operation, the patient and the surgeon both profit, as technically the task is much easier. In cases in which the prostate is very vascular, a supplementary transfusion should be given if the loss of blood is at all excessive. A few extra transfusions of blood will be well spent in an effort to guard against the distressing complications mentioned earlier in this paragraph.

Late bleeding may occur at any time but rarely occurs in any great amounts after fourteen days. With the slough incidental to the healing process, bleeding occasionally will occur after the patient has been dismissed. Rest, plus the forcing of fluids, will usually clear the complaint. In some instances lavage of the bladder or even an indwelling catheter for twenty-four to forty-eight hours may be necessary. When there is considerable inflammation along the prostatic mucosa and small amounts of bleeding occur occasionally, urinary antiseptics and particularly methylene blue may be of help. The latter drug is helpful because of its mild hemostatic properties and also because of its ability to hide the minute bleeding which may cause the apprehensive patient much concern.

Inadequate removal of tissue may be responsible for continued hemorrhage, and when the latter occurs over a period, cystoscopic examination is indicated. Residual tissue or regrowth of tissue frequently explains hemorrhage. Infection also may be the cause of continued hemorrhage.

DYSURIA

In a previous discussion of this paper, I called attention to the burning, frequent urination, nocturia, posterior urethral distress, painful voiding and pain in the perineum, which occasionally occur postoperatively. When these symptoms are present immediately upon removal of the catheter after operation, I do not feel that the surgeon should be greatly concerned. A raw sloughing area in the prostatic bed, washed with urine, is bound to be sore. When the symptoms persist for any period, one must attempt to find the cause. Infection undoubtedly is the major causative factor for these symptoms either alone or collectively. Such being the case, one might feel that urinary antiseptics should be given prophylactically, but I do not believe that this is good practice. In the first place, elderly men do not tolerate the sulfonamide compounds well, and secondly, in the presence of the large, raw area in the prostatic urethra, sterilization of the urine is well-nigh impossible. If the infection is great, lavage of the bladder and chemotherapy by mouth may serve to reduce the amount. If a sulfonamide compound is used, it should be given in small dosages, never exceeding 45 grains (3 gm.) daily for a period of one week, and the intake of fluid should not be less than 2,500 c.c. daily.

A piece of tissue floating free in the bladder or attached by a small tag of tissue, or the inadequate removal of tissue, may produce posterior urethral distress and dysuria. If local measures such as lavage of the bladder and instillations along with chemotherapy do not relieve these symptoms, cystoscopic examination should be advised.

Frequency and nocturia are more often due to a large fluid intake than to any other cause. Limiting fluids to eight glasses daily, with instruction that this should be taken mostly before 4 p. m., will often help. Nocturia among the aged occurs often and, I feel, in many instances is not directly attributable to prostatic difficulty in the postoperative case. Many of these patients are poor sleepers, and, as we all know, awakening at night for any reason makes us aware of an urge to void. Another observation has been made that older persons secrete more urine at night than younger persons, perhaps owing to the fact that rest in bed tends to improve their circulation, and hence a greater amount of blood passes through the kidneys with a resulting increase of urinary secretion.

One seldom encounters perineal distress postoperatively, but if persistent it may be relieved by roentgen treatment of the perineum. This distress may accompany dysuria after resection with the electric loop in cases in which fulguration has been too deep. Local instillations may help but usually time is required before this distressing symptom disappears. Prostatic massage is seldom indicated and I do not believe that it should be used within six to eight weeks of the operation.

Residual Urine

In general, inadequate removal of tissue, infection, atonicity of the bladder, and diverticulum are the usual causes for the continuance of residual urine. In the absence of the latter three causes, persistent residual urine suggests the need of cystoscopic examination and residual tissue frequently will be found. Existing infection may produce enough irritation and edema to cause residual urine and, if present, should be eradicated.

The frequency of performance of diverticulectomy is decreasing and this is undoubtedly due to our experience with diverticula in relation to transurethral prostatic resection. During the last few years a large group of patients who have had large diverticula of the bladder have undergone prostatic operations at the clinic. In almost all cases the diverticulum has not required any further surgical treatment and eventually has shrunk to an imperceptible lesion if the tissue at the vesical neck was resected adequately and an adequate channel provided. Frequently, daily catheterization to drain the diverticulum has been needed postoperatively in order to enhance its shrinking.

When one has a patient with a large atonic bladder incidental to long-standing obstruction, provision of an adequate channel through the prostatic urethra will not relieve residual urine. The atonicity is no doubt responsible and if one resorts to regular intermittent catheterization, vesical tone may be restored with the eradication of residual urine. Such a procedure may be necessary for a period of weeks.

In the fibrotic type of prostate, the reformation of scar with dysectasia and spasm of the vesical neck may occur, and if present, dilatation should be resorted to. In some of these cases further resection may be indicated. This particular group is undoubtedly the most difficult to treat.

Incontinence

Incontinence is a rare complication and need not occur. When present in cases of resection of benign hyperplastic prostatic tissue, it is almost always due to faulty technic of the surgeon. The surgeon must be familiar with his landmarks and learn to stay well within the external sphincter. He must use care in manipulation of the instrument, as frequently trauma to the sphincter may occur as a result of evulsion of the fibers rather than of actual cutting of the sphincter. In such cases, the incontinence eventually disappears and this can be helped by instructing patients to exercise the muscles of the perineum by regular voluntary contractions. Unfortunately, with malignant lesions of the prostate the sphincter frequently is invaded by carcinoma and its muscular contraction destroyed. When this occurs, a penile clamp is most helpful. These clamps are not the most comfortable things in the world to put on without the proper adjustment. A little care on the part of the surgeon and co-operation on the part of the patient will convince the patient that the urethral clamp is a real friend.

Sexual Disturbances

Sexual disturbances are not common and come without any degree of regularity. Usually there is no change of libido or potentia but in some cases these are increased and in others decreased. The most frequent complaint is the loss of ability to ejaculate.

This is the result of anatomic factors. As the region of the vesical neck is opened widely at the time of operation, it is easier for the ejaculated semen to pass into the bladder than out of the urethra. A few words of reassurance are again all that is necessary to help the patient and put his mind at ease.

COMPLICATIONS

Epididymitis may occur but usually can be avoided if the patient is instructed not to let his bladder get too full, not to strain and not to do any heavy work for three or four weeks after operation. When epididymitis does develop, adequate support to the scrotum, rest, warm sitz baths and chemotherapy usually will clear up the condition. Occasionally an abscess may form and, if so, incision and drainage are necessary.

Prostatic abscesses are rare. Thrombophlebitis of the veins of the legs may occur and, if it does appear, should be treated as usual. Rest, elevation of the extremity and application of hot moist packs are indicated. This complication may be followed by fragmentation of the clot with secondary emboli reaching the lung and producing infarction. In rare cases death ensues.

Headaches may occur after spinal puncture but these usually can be relieved by rest and disappear within a few days.

COMMENT

As one reconsiders the points that I have discussed there is one outstanding fact which contributes in large measure to the complaints we all encounter after transurethral resection of the prostate. This is technical inadequacy on the part of the surgeon. The operation requires special skill and special training and unless the surgeon has these his patients will besiege him postoperatively with all sort of complaints. Fortunately only a small percentage of patients experience any appreciable distress after operation, and with more experience and greater care on the part of the surgeon I am certain this percentage can be reduced still further.

Through this paper I have had much to say about technical surgical inadequacy. In fairness to an effective procedure and to those of my own branch of the profession however, and even at the expense of repetition, I wish to close with the following declarations: Although the procedure is not to be considered a simple one, the percentage of satisfactory results achieved by experienced surgeons is as high as, or higher than, that attained with any other method of prostatectomy. The morbidity and mortality are lower. I already have said what may be expected of the future.

The Management of Eclamptogenic Toxemia

By FREDERICK H. FALLS, M. D.
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The management of eclamptogenic toxemia depends upon many factors which differ in different cases. Chief among these are (1) the time during pregnancy that the symptoms of the disease appear; (2) the rapidity of their development with or without treatment; (3) whether the patient is a primipara or a multipara; (4) the surroundings and the technical skill of the physician. This malady has been called the disease of theories because various explanations have been advanced from time to time by different authorities as to the etiology, none of which have explained satisfactorily all of the clinical facts associated with the disease.

However, if we are to treat these patients intelligently it is necessary to construct a theory as to what factors are at work to produce the lesions and symptoms of the disease, in order to eliminate some and minimize the harmful effect of others. It is not necessary to conceive of this theory as one hundred per cent correct in all details. Its usefulness will be vindicated if on application it works to reduce the mortality and morbidity caused by the disease, which is the second most frequent cause of death among pregnant women.

Many older writers on this subject are so vague in stating their conception of the etiology and pathology of the disease that one feels a cloak of mystery surrounding the facts in the case which is difficult to penetrate and arrive at a logical basis for a rational treatment.

For this reason I wish to submit to you a simple diagram which I have used for years in the teaching of undergraduates to present the principles of the pathogenesis of the disease. We will then discuss our method of treatment based on this conception and report to you the results of our observations over a period of years. We have not varied this treatment to any considerable extent for the obvious reason that we wished to be able to compare our results with those of others using similar and dissimilar methods in order to evaluate its effectiveness.

Let us start out by stating that the pregnant woman, because of the demands of the fetus, must of necessity have a marked change in many important bodily functions, especially metabolism and the elimination of the end products of metabolism. Of the latter the most significant for our consideration, because they are the most toxic, are the end products of protein metabolism. In early pregnancy because of the increased activity of the thyroid as shown by the increased basal metabolic rate an increase in these products is to be expected in all pregnant women. We may assume that the excretory organs are capable of handling this increase satisfactorily in almost all cases, and that no clinical signs or symptoms point to the fact that additional strain is placed on these organs. However, it has long been known that if carefully examined, the urine of pregnant women will, in most cases, show a slight albuminuria and a few casts and red cells are very frequently seen. Further, we have shown that carefully run phenolsulphonephthalein tests show a decrease in the functional capacity of the kidney in otherwise normal pregnant women of approximately 30 per cent.

It is fair to conclude then that every pregnant woman is mildly toxic because of the increase of toxic end products caused by the increased metabolic rate in the early months of pregnancy, augmented later in pregnancy by the additional burden of the end products of fetal metabolism that are poured into the maternal blood stream, and must be eliminated by the maternal excretory mechanism. When this mechanism fails to meet the challenge of the increased burden for any reason, an eclamptogenic toxemia develops. It will be seen therefore, that the control of this disease lies in the ability of the woman to maintain a balance between the amount of toxins getting into her blood and the amount she can get rid of. The management of the condition, then reduced to simplest terms, depends on decreasing the intake of toxins and increasing their output, especially until the fetus has passed the age of viability when if necessary, the pregnancy may be terminated without sacrificing the baby.

The circulation of these toxins gives rise to certain changes in several vital organs which may be well to point out. They are liver, kidney, brain, heart muscle, retina and skin.

It is important to remember that the treatment of the lesions in these organs is primarily a matter of eliminating the original source of the lesions, the fetus and placenta. Also, that even after these have been removed the damage produced in these organs continues to exert a depressing influence on their functional capacity to excrete these toxins which keeps the patient in jeopardy until the reparative changes in the kidney, liver, heart and brain have had time to take place.

The cases that present themselves may be divided into several groups.

Group I. Mild toxemia, slowly developing, are treated by bed rest at home or in the hospital. Milk diet, daily magnesium sulphate, daily blood pressure and urine analysis — quantative albumin, phenolsulphonephthalein, blood count and hemoglobin, and weight observations.

If improvement occurs, increase diet and allow more activity. Blood pressure, weight and urine analysis should be estimated twice a week. If on the other hand no improvement occurs after this treatment has been applied for a week, the termination of pregnancy must be seriously considered because of the danger of convulsive seizures occurring. This is the group which furnishes the real opportunity to reduce maternal and infant mortality by applying the principles of preventive medicine, that is, good prenatal care, and, assuming good cooperation by the patient, should furnish the proof that at last we are beginning to control the disease.

Group II. This group of patients comprise those who have had a mild toxemia but have failed to respond to treatment or who showed severe symptoms from the start, and also represents that group on whom palliative measures have failed to make any favorable impression after forty-eight hours.

This group contains more severe cases as a rule. They furnish a fairly large number of fatal cases. In our experience palliative management has very limited usefulness. A few days may be thus used to confirm the impression that these cases cannot be managed by the same means which yields success in such a large percentage of Group I cases. Furthermore, after forty-eight hours of palliative management the patients are better prepared to withstand the strain of induced labor or the operative delivery which may be necessary in most of the cases. To bring about delivery, bag induction may be used. rupture of the membranes in suitable cases or cesarean section. Depending upon the conditions present in the individual case.

Group III. Group III cases are the so-called fulminating cases. These patients suddenly develop severe symptoms which increase in intensity almost by the hour. The convulsive seizures appear early, are extremely violent and succeed one another in rapid succession. No palliative treatment can be relied upon in these cases to give good results.

The termination of the pregnancy by the most conservative method is the procedure of choice. Usually the condition of the patient at the time the diagnosis is made is such that a quick cesarean section, low cervical or classical will give the best results if hospital facilities and a competent surgeon are available. If not, the Strogonoff treatment is recommended with the understanding that it is not quite as safe for the mother and definitely less so for the baby. In this connection it may be stated that a mixture of these methods is undesirable. If one is forced to choose the Strogonoff method through force of circumstances, it should be entered into and carried through. A late operative delivery after numerous convulsions and after the patient's condition has deteriorated is highly dangerous. Such procedure has led to disaster and to the unfavorable attitude toward operative intervention in eclampsia. These cases call for immediate decision and prompt action, and only on this basis can favorable results be expected. The details of the administration of the Strogonoff treatment are as follows:

1. Morphine sulphate grains ¼ hypodermically as soon as patient is seen.
2. One hour later 20 - 40 grains chloral hydrate per rectum.
3. Two hours later morphine sulphate ¼ grain hypodermically.
4. Four hours later 30 grains chloral hydrate per rectum.
5. Six hours later 15 - 20 grains of chloral hydrate per rectum.
6. Seven hours later 20 grains of chloral hydrate per rectum.

Group IV. Intrapartum eclamptic convulsions present widely different problems according to the stage of labor and the progress made at the time the patient comes under observation. If labor has just started and the patient is a primipara with only slight cervical dilatation, a cesarean section is the method of choice for delivery if favorable surroundings and an able surgeon and assistants are available. Failing these requisites, resort must be made to the conservative Strogonoff management. If on the other hand the cervix is dilated fifty per cent or more before the first convulsion, sedatives should be given until complete or near complete dilatation at which time Dührssen's incisions at 10 — 2 and 6 may be made, to complete dilatation, followed by version and extraction or forceps delivery, depending on how deeply the presenting part is engaged, and whether or not labor seems to be delayed after completing the first stage. If on the contrary the presenting part is at or near the perineum when the first convulsion occurs, even when the cervix is only partially dilated, labor will terminate rapidly in most cases spontaneously, and the chief concern of the attendent should be to provide sufficient help to avoid contamination leading to subsequent puerperal sepsis.

For this reason such patients should be under close observation and the progress of labor frequently evaluated, in the first place to avoid, if possible, precipitate delivery during a convulsion, and in the second place, to postpone putting on the sterile drapes too soon to avoid their contamination by numerous convulsions. It is our practice when facilities are available as in hospital practice, to deliver these cases with outlet forceps and local anesthesia of the perineum between the seizures.

As soon as any patient has delivered who has manifest signs of preeclamptic toxemia, except of the mildest type, we treat them prophylactically in the following manner: Morphine sulphate gr. ¼ or 1/6 depending on the size of the patient, to reduce the irritability of the central nervous system. Magnesium sulphage oz. 1 A. M. and P. M. until watery stools are obtained. Fluids are forced and if the blood pressure rises or remains high a venasection of 500 to 700 cc. is done if convulsions occur or seem imminent.

By anticipating the possibility of convulsive seizures in this way their occurence and severity are reduced to a minimum. This management is continued until the urine begins to clear of albumin, the blood pressure falls, and the phenolsulphonephthalein test shows an excretion of fifty per cent or more.

It is necessary of course to protect the patient against injury during a convulsion, but there is another prophylactic measure often neglected in the average case that should be observed, namely, the protection of the baby. Until the danger of postpartum convulsions is definitely past the baby should not be put to the breast because of possible injury by the mother. Furthermore, these patients should be carefully observed for evidence of mental aberration as injury to the baby might result from this cause.

The management of the eclamptic convulsion itself can be summed up in the word protection. Protection from outside stimuli such as noises, unnecessary examinations and operative deliveries will reduce the incidence and number of convulsions. The tongue can best be protected by use of a mouth gag, made of a wrapped clothespin. Constant attendance by a trained person is necessary, especially when the patient is being transported on a cart. Oxygen should be available after the convulsion to reduce the harmful effects of asphyxia and a suction apparatus should be available to clear the air passages when necessary.

TREATMENT OF THE BABY

In the discussion of the treatment of eclamptogenic toxemia very little is said about the effect of the treatment on the welfare of the baby. Since in all pregnancies two patients not one must be safeguarded, this is a serious mistake. Several factors enter into the equasion which produce unfavorable conditions for the baby. The toxins circulating in the maternal blood have a deleterious effect on the nutrition and development of the fetus, especially if the toxemia of the mother has been prolonged and severe. The circulatory disturbances in the placenta also reduces the nutrition and oxygen supply of those babies and a normal development could hardly be anticipated under the circumstances. The time of onset during pregnancy (usually about the sixth to eighth month) leading as it frequently does to spontaneous or induced premature labor still further jeopardizes the chances of the baby surviving in extrauterine life. Another factor which increases the hazards of these babies is that operative intervention so frequently is resorted to in the interest of the mother or because of threatened fetal asphyxia.

Of the various important causes of fetal mortality in the eclamptogenic toxemia case premature detachment of the placenta ranks high. Hemorrhagic changes in the body of the placenta may also be sufficiently extensive to result in fetal death. For this reason it is necessary in any case in which the fetus has attained the age of viability to watch for evidence of fetal asphyxia. An increase of the fetal heart rate to 170 or above if sustained or if followed by a drop below 100 per minute, irregular and weak indicates that the baby in most cases is in danger. Removal, therefore, as soon as practical is indicated. The means to this end vary with the circumstances surrounding the case. If not in labor, a cesarean section should be done. If well advanced in labor incise cervix if necessary to complete the dilatation and deliver by forceps or version, depending on the degree of descent of the head at the time the delivery becomes necessary.

Forceps, version, breech extraction and cesarean section all take their toll in fetal mortality, both at the time of birth of the baby or in the days immediately after delivery. For these reasons in every case the condition of the baby and its chances of survival must be given due weight by the attending physician when pondering the various factors entering into the decision as to what constitutes proper management in a given case.

Because in many instances it seems best to terminate pregnancy before the fetus is fully developed in spite of the risks involved, it will be necessary to safeguard the newborn by throwing around it all the protective devices at our disposal. A heat bed, incubator, tracheal catheter, oxygen, special nursing care, breast milk, gavage feeding, plasma and whole blood, all should be available for use if necessary, at the time of delivery, and not assembled hurriedly after the birth of the baby. A few minutes of good care on the delivery table and for the first few hours thereafter is worth more than days of careful handling after the transition from intrauterine to extrauterine life is several hours in the background. Because this is so, it is of importance to see that adequate help is on hand to take over the care of the baby, if this is necessitated by the preoccupation of the attending physician with the management of the mother if she is in serious condition.

The value of any form of management must be judged by the results obtained by its use. The significance of the figures depend upon the number of cases and to some extent on the time interval over which they have been collected. Almost any management of this disease might show good results in a small number of cases over a short space of time. This series comprises 1200 cases collected over a period of twenty-one years. There has been no significant change in the management throughout this period of time. With the development of intravenous glucose solution therapy in various conditions a few of these cases have had some glucose intravenously. The results are not strikingly different in these cases than in the majority of the cases not so treated. Likewise, with the advent of intravenous administration of magnesium sulphate some of the cases were given this form of therapy instead of confining ourselves to oral administration. No great improvement in the results were noted, although it is my clinical impression that possibly the improvement in the condition of the patient was accelerated by the intravenous use of the drug.

The maternal mortality of 8 represents a total mortality of all cases who died in whom a diagnosis of preeclamptic or nephritic toxemia had been made. This brings the rate for these cases down almost to the level for normal pregnancy.

We had a gross fetal mortality of 11.9 per cent which we feel is good when we consider the prematurity and other disadvantages under which a large proportion of these infants were born. In spite of what we have said about our conviction of the necessity of cesarean section the incidence of 4.2 per cent for the entire series shows a conservative attitude toward the use of the operation except for special indications. The low cervical operation was done 15 times and the classical 8, while one Porro operation was done. Forceps were applied in 6.2 per cent of cases and version was done in 2 per cent of the cases. This leaves a total of 1131 cases that were delivered spontaneously. By this management only 50 of the 1200 cases developed convulsions.

We believe that these results can be duplicated or even bettered by any well regulated maternity hospital or private physician who will take the trouble to apply the principles laid down in this communication.

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